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EFFECT OF RADIUM EMANATIONS ON THE LARYNGEAL CARTILAGE

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We realize full well the difficulties encountered when one is called on to prescribe treatment for malignant disease in the region of the tonsil, hypopharynx or larynx. Likewise we are aware that treatment in any form thus far developed has proved inadequate in a lamentably high percentage of cases.

In reviewing the list of remedies available, we find that it consists of surgical removal, destruction by heat, destruction by irradiation or a combination of surgical removal and irradiation.

The treatment of this type of disease is attended by difficulties peculiar to the region in which it is located in that the neoplasm, because of its inaccessibility, is not readily visible and therefore is usually seen in a late stage. Even when it is seen early, the tendency of this type of tumor to metastasize early and the fact that the lymphatics are practically always involved further render a successful result of treatment unlikely. Additional difficulties arise because of the almost certain interference with respiration and deglutition and because of the danger of serious injury to tissues such as the laryngeal cartilages, the thyroid gland and the great vessels.[1]

With the advent of radiation therapy, the possibilities for its use in this field were hailed with delight, but experience has shown that its application is attended by serious difficulties. The literature abounds with reports of clinicians throughout the world who are well entitled

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1. Mackenty, E. J.: Surgical Treatment of Laryngeal Cancer, New York State J. Med. **22**:456 (Oct.) 1922. Iglauer, S.: X-Ray Injury to the Larynx, Ann. Otol., Rhin. & Laryng. **36**:124, 1927. Klestadt and Fürst: Pathologisch-anatomischer Beitrag zur Röntgenspätschädigung des Kehlkopfes, Ztschr. f. Hals-, Nazen- u. Ohrenh. **31**:281, 1932. Lynch, R. C.: Cancer of the Larynx, Tr. Indiana Acad. Ophth. & Otolaryng., 1926, p. 12.

to an opinion on the subject wherein they describe results so discouraging as to constitute a contraindication for the use of this method.

However, within the past few years, as a result of increased knowledge of the entire subject of radium, its emanation, roentgen radiation and methods of application, as well as an increased knowledge of tumors in general, we have renewed hope as to the possibilities for the use of this remedy.

We are of course impartial as to the remedial agent to be applied, our main desire being to find one, or a combination of several, which will lead to complete destruction of the neoplasm without serious danger to the neighboring tissues. In practice, the fact remains that in many cases irradiation is the only method left open for consideration, and it is debatable whether or not it should be employed in those cases in which surgical removal is carried out.

In view of the recent developments in the field of radiology, many of the clinicians [2] referred to have modified somewhat their opinion regarding the possibility of its use.

The work described in this paper is based on the belief and hope that radium emanations may be employed in and near cartilage without its destruction, and the reports of cases which have a bearing on the subject are included.

In reviewing the literature, we find reports of the effect of radium and its emanations on every tissue of the body save cartilage, and the remarks of some of the leading clinicians are incorporated herewith as a matter of reference.

The trend of progress is critically described by Thomson and Colledge.[3] They stated:

> Usually the tumour only shrinks temporarily. If any enlarged glands are present, they do not disappear, and there commonly results damage to the healthy tissues with necrosis of the laryngeal cartilages. Bone absorbs larger quantities of radiant energy than other tissues on account of the high density and atomic weight of its constituents, and the same holds good for ossified cartilage. This high absorption of energy causes intense secondary radiation, with resulting destruction of living cells, and any secondary infection produces necrosis of cartilage especially in aged persons. . . . Methods in consequence are still changing, but a point has been reached at which they have become to some extent standardized and have given definite results. . . . It appears that some of this failure has been the result of incorrect, usually excessive doses, misapplied.

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2. (*a*) Cutler, M.: Comparison of the Effect of Unfiltered and Filtered Tubes Buried in Rabbit Muscle, Am. J. Roentgenol. **16**:535, 1926. (*b*) Quimby, Edith: Comparison of Different Metallic Filters Used in Radium Therapy, ibid. **13**:330, 1925; (*c*) Intensity of Radiation in the Vicinity of Filtered Radon Implants, Radiology **10**:365, 1928.

3. Thomson, St. Clair, and Colledge, L.: Cancer of the Larynx, New York, The Macmillan Company, 1930.

Desjardins,[4] in his series of articles on the influence of irradiation on the tissues of the human body, commented on the fact that "each variety of cell in the body has a specific sensitiveness, or rather a specific range of sensitiveness, to irradiation." He continued: "The specific sensitiveness of each kind of cell looms up as the dominant single fact of radiology and deserves to be recognized as a law." He said that "if one may judge by present day writings, the existence of such a law and of its medical and biological implication is not at all realized." [5] He then described in detail the changes effected on practically all the tissues of the body save cartilage. Concerning this tissue, he said that no definite statements regarding its reaction to irradiation have been made. Broders' [6] classification and its application from the standpoint of radiosensitivity are most interesting, but we have found to our disappointment that tissues do not always behave as they would be expected to do according to this theory.

Souttar [7] has written an excellent article which deals with the physical characteristics of radium emanation, dosage, methods of application and clinical results. His article is helpful to those wishing to study the fundamentals of the preparation of radium and its emanations. In his discussion of types of containers, he expressed a preference for platinum as the most effective screen, but he added that silver has worked very well for him, especially when it is planted in the tissue. On the subject of dosage, he wrote:

> It is a generally accepted standard of dosage that one millicurie of radon during its decay will produce a Gamma radiation sufficient to destroy all carcinoma cells within a distance of 0.5 cm. This is, of course, a very rough estimate, for no account is taken of the considerable variation in the sensitivity of the various types of cancer.

Failla,[8] Quimby,[2b] Quick [9] and others described in detail the various types of containers for the emanation of radium as well as of its salts. Failla treated of the development of the gold seed in detail and discussed the advantages of this type of container as compared with platinum needles, glass seeds and other forms of applicators. Quick gave an interesting and instructive historical review of the use of

4. Desjardins, A. U.: Action of X-Rays and Radium on Heart and Lungs, Am. J. Roentgenol. **28**:75, 1932.

5. Desjardins, A. U.: Radiosensitiveness of Cells and Tissue, and Some Medical Implications, Arch. Surg. **25**:926 (Nov.) 1932.

6. Broders, A. C.: Carcinoma in Situ Contrasted with Benign Penetrating Epithelium, J. A. M. A. **99**:1670 (Nov. 12) 1932.

7. Souttar, H. S.: Observations on Use of Radon Seeds in Treatment of Carcinoma, Brit. M. J. **2**:273 (Aug. 23) 1930.

8. Failla, G.: The Development of Filtered Radon Implants, Am. J. Roentgenol. **16**:507, 1926.

9. Quick, D.: Radium in Cancer Therapy, Brit. M. J. **2**:765 (Nov. 8) 1930.

radium up to 1930, calling attention to the work of Failla, who replaced the unfiltered radon seeds by a gold capillary tube. He enumerated the many advantages of the gold seed over other types of container. He remarked that the comparative ease with which the shorter gamma rays can be obtained from radium makes it superior to roentgen radiation.

It seems to be generally thought, and it is our feeling, that gold seeds possess advantages over all other containers for applying emanations of radium. Certainly the gold seed can be employed in almost any region of the body without fear of infection. It does not require removal later, and the amount of emanation may be measured. Crossfire can be fairly well controlled by accurate placing. Duffy [10] and other workers in this field have demonstrated that most of the beta rays will undergo decay within the gold seed, whereas the shorter gamma rays pass through the wall of the seed into the tissues. In view of the comparatively infinitesimal amount of gamma rays required to kill cancer cells within their immediate vicinity, the advantages of this type of applicator seem apparent. Attention is directed to the fact that, as with the roentgen rays, one is dealing with a remedy which is not without danger. Soiland and Costolow [11] expressed the feeling of many workers when they pointed out the great danger attendant on the application of this remedy, particularly when it is employed by those considered incompetent or inexperienced in its use. Although enthusiastically in favor of the use of such implants, especially where the blood supply is good, they realized the possibility of damage to normal tissue as a result of faulty placing and made a plea for cooperation between the radiologist and the clinician, saying:

> In the face of much adverse criticism the writers call attention to the altogether too prevalent use of radium implants by general practitioners, urologists, gynecologists, and laryngologists in cases which require the best skill and knowledge of trained radiologists. This is one of the outstanding reasons for the confusion and controversies of which radiation therapy is the storm center. . . . It is not intended by this statement to convey the idea that all radiologists can with ease . . . make accurate insertions of radon seeds. . . . Far from it! In all such cases, the combined skill of the surgeon and the radiologist must be correlated in order that the patient may obtain the best end result possible at the present state of knowledge.

The following cases are thought pertinent to the subject.

REPORT OF CASES

Case 1.—J. R., aged 54, white, a widow, was first seen in 1929 for an intractable cough, which she had had for years. Bronchoscopic examination elsewhere by

10. Duffy, J. J.: Use of Gold Filtered Radon Seeds, Radiology **12**:93, 1929.

11. Soiland, A., and Costolow, W. E.: Comparative Notes on Radon Implants, Radiology **12**:45, 1929.

competent observers failed to demonstrate the cause for the cough. After study by numerous internists and others, the cough was still unexplained. There was gradual loss of weight. The patient disappeared from Dr. Arbuckle's service and was not seen again until 1932. At that time she had a history of wheezing in 1931.

On April 14, 1932, she coughed up blood and a fragment of tissue which microscopic examination proved to be squamous cell carcinoma. Bronchoscopy on May 1 disclosed a tumor on the wall of the trachea at about the level of the second and third rings on the right side. On May 14 the tumor was about 1 inch (2.5 cm.) in diameter and stood up from the surface of the trachea about ⅛ inch (0.3 cm.). It appears that in November 1931 a diagnosis of chronic tracheitis was made, and high voltage roentgen therapy was applied to the lymphoid tissue in the pharynx. On May 15 this treatment was repeated, and on May 24 four gold seeds each containing 0.5 millicurie were planted endoscopically in the base of the tumor. On June 21 the size of the tumor was much diminished. On July 5 one gold seed was implanted in a suspicious-looking area, but at this time no tumor was visible.

The patient's condition improved with a summer in the North, but pain in the region of the sternum and in the vertebral column gradually developed. There was a slight redness at the site of the original lesion and two seeds of 0.5 millicurie each were implanted. Roentgenograms at this time showed a destructive process in the fifth dorsal vertebra, and on December 4 roentgenograms of the lung showed masses suggestive of metastasis throughout both lungs. On Feb. 15, 1933, there were definite metastatic nodules with multiple abscesses throughout both lungs, from which the patient died on July 6.

Case 2.—J. O., aged 63, an Austrian-born coal miner, who came to the United States in 1895, entered the medical service of the Barnes Hospital on Sept. 16, 1932, because of severe cough, a moderate amount of mucopurulent sputum and shortness of breath of about two months' duration. Repeated inquiry placed the beginning of the present illness two years earlier, with the onset of a dry cough and slight wheezing on exertion. The patient's appetite had been poor for three months, and he had lost considerable weight.

Important physical findings were limited to the chest, aside from a poor state of nutrition. There were moderate emphysematous changes. The lower portion of the right side of the chest presented anteriorly and posteriorly limitation of expansion, dulness to percussion, increased tactile fremitus and harsh breath sounds. Roentgenographic studies showed an atelectatic middle and lower lobe of the right lung, displacement of the heart to the right and a triangular area of increased density extending from the hilus outward and downward to the right costophrenic angle. Roentgenograms of the chest made following the injection of iodized poppy-seed oil 40 per cent showed narrowing of the bronchus of the lower lobe of the right lung.

For two weeks the temperature curve was septic, varying from 37 to 39 C. (98.6 to 102.2 F.). A provisional diagnosis of abscess of the lung and probable malignant growth was made. There was no improvement, and bronchoscopy was requested. On September 30 the bronchus of the lower lobe of the right lung was found to be blocked just below the orifice of the bronchus of the middle lobe. The narrowed lumen was obstructed by a plug of débris. When this was removed, it was possible to traverse a narrowed bronchus for several centimeters, to enter a large abscess cavity and obtain a large quantity of pus. Iodized oil injected at bronchoscopy showed a fluid level near the wall of the right side of the chest when the patient lay on his left side.

Pus from the abscess yielded a growth of Staphylococcus albus, hemolytic streptococcus, Micrococcus catarrhalis and Bacillus influenzae. Inoculation of a

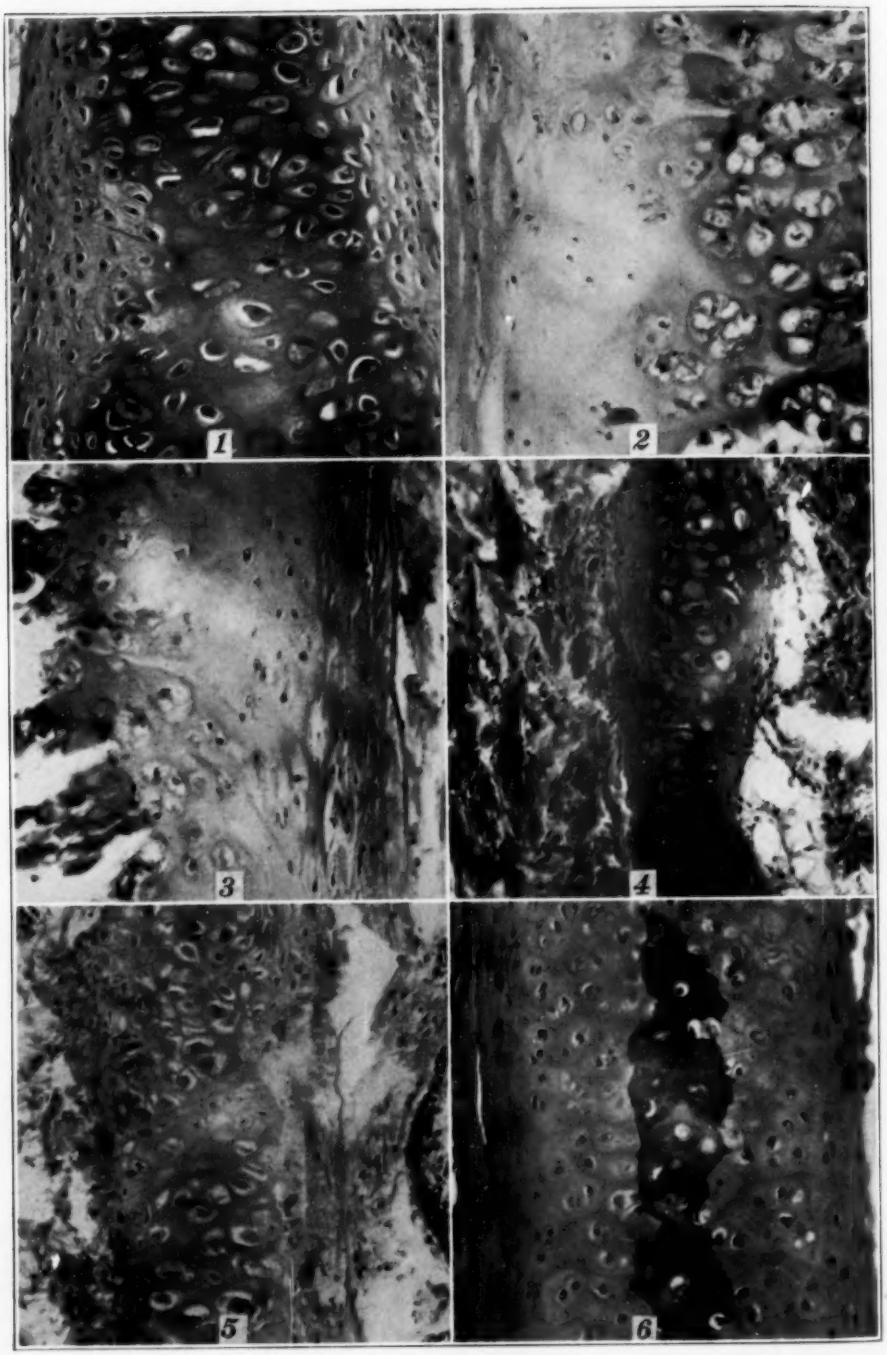

Figure 1

EXPLANATION OF FIGURE 1

Fig. 1.—Sections of a laryngeal cartilage from dogs showing the effect of implanted gold radon seeds. All photomicrographs in both figures were made at a magnification of 140 diameters by Dr. Gordon H. Scott. The direction of radiation from the seeds is in all cases from left to right, with the exceptions noted in the text. Section *1*, from dog 4, given 665 millicurie hours of radiation, the seed being 0.5 mm. from the cartilage, shows the near and remote surfaces of the cartilage, and there is no detectable difference in the cells or intercellular substance. Section *2* is from another block of tissue from the same dog. The seed was 1.25 mm. from the cartilage. The near surface of the cartilage is illustrated and differs from the remote surface (section *3*) by (1) a reduction in the number of nuclei of the perichondrium, (2) swelling of the intercellular substance as if water had been absorbed and (3) an increase in the affinity for eosin. Section *3* shows the remote surface of the cartilage in the block tissue used for section *2*. Section *4* is from dog 5, which likewise received 66.5 millicurie hours of radiation, the seed being 3.75 mm. from the cartilage. The cartilage consisted of sections of two laminae separated by a core of fibrous and fatty tissue. The lamina nearest the seed is shown. It is much more acidophilic than the distant one (section *5*). Section *5* shows the distant lamina. Section *6* is from another block of tissue from dog 5, the seed being 0.4 mm. from the cartilage. The nuclei of the fibroblasts and cells of the perichondrium near the seed are more deeply stained than the remote ones. Calcification is beginning in the middle of the cartilage, as indicated by an irregular stratum of intensely stained (basophilic) material.

guinea-pig gave negative results for tuberculosis. The day after bronchoscopy the cough was less, and the patient felt much better.

Bronchoscopic drainage was repeated at weekly intervals during October, the inflammatory reaction in the bronchus of the lower lobe of the right lung gradually decreased, and the septic temperature subsided. On October 28 there was visible tumor-like tissue in the constricted area of the bronchus, and biopsy showed squamous cell carcinoma.

On November 16 four gold radon seeds of 0.5 millicurie each were implanted in the mass in the wall of the bronchus of the lower lobe at 5 : 30 and 7 : 30 o'clock; at 6, 1 cm. above, and at 2 : 30, 0.5 cm. lower. On November 25 a diagnosis of encysted empyema was made, and three days later thoracostomy was done for drainage. On November 30, while sitting up in bed eating supper, the patient suddenly fell back dead.

Permission for autopsy was not obtained, but some tissue was removed by bronchoscopy post mortem. This included some sloughed-off cartilage, a section of which is illustrated in figure 2, *11*. As one would expect, most of the cells were necrotic and had lost all traces of nuclei. The size of the tumor mass within the bronchus had been reduced by about one half.

Case 3.—J. B., aged 48, white, an iron molder, entered the Barnes Hospital from the outpatient clinic on Dec. 13, 1931. The chief complaint was general weakness and loss of appetite, which had begun about twenty-six months before. For the last sixteen months there had been a persistent cough with a thick, clear, odorless sputum. For the last four months the patient had a dull pain in the right infraclavicular region, with night sweats and dyspnea. He had lost 30 pounds (13.6 Kg.) in weight. He had pleurisy nineteen years before.

Physical examination disclosed dulness and faint tubular breathing over the upper portion of the right side of the chest with râles and inspiratory wheeze of the right side. There was paresis of the right vocal cord. The sputum was blood-streaked; no tubercle bacilli could be found. Roentgenograms showed an area of greatly increased density over the field of the upper portion of the right lung; there were no bone changes.

Diagnostic bronchoscopy on December 17 demonstrated a carcinoma obstructing the bronchus of the upper lobe of the right lung and extending into the main bronchus. Subsequent examination demonstrated that this tumor was of rapid growth and extended down the main bronchus. On Jan. 4, 1932, six gold seeds of 1.5 millicuries were implanted bronchoscopically. On January 19, or fifteen days later, the endobronchial tumor appeared to be about half its former size, and on January 29 it had almost entirely receded from the main bronchus. On February 9, thirty-five days later, the only remaining sign of the malignant disease in the bronchus was a slight thickening on the posterior wall just below the opening of the bronchus of the upper lobe. The upper lobe remained atelectatic, however, and on February 15 thoracostomy was done, many adhesions and numerous small abscesses being found in this lobe. Cautery-pneumonectomy was carried on in these cavities. Seeds were implanted, and high voltage roentgen therapy was given later. On April 15, during an attack of severe coughing, the wall of one of the abscess cavities was torn, and there was hemorrhage sufficient to require blood transfusion. Nine or ten days later the patient died of bronchopneumonia.

At autopsy, metastases to the mediastinal glands, the liver and the kidneys, as well as bronchopneumonia and an abscess in the middle lobe of the right lung, were revealed. There was no gross evidence of tumor in the main bronchus. A block of tissue containing two radon seeds was removed under fluoroscopic vision

for microscopic study. Figure 2, *10*, shows shrinkage of cells, pyknosis of the nuclei and acidophilia of the ground substance.

CASE 4.—J. D., aged 65, a night-watchman, entered the Barnes Hospital in Dr. L. W. Dean's service through the dispensary on Nov. 6, 1930, for study of a lesion of the throat. The chief complaint was that about six weeks previously he had difficulty in swallowing solids and liquids, coughed considerably, was hoarse and had difficulty with breathing. There was no loss in weight. His personal and his family history were essentially unimportant.

Examination revealed a mass about 1.5 cm. in diameter involving the rim of the larynx on the right side and extending to the base of the epiglottis and out into the pyriform sinus. The right arytenoid was fixed. The mass was ulcerated. There was a firm gland in the anterior triangle of the neck on the right side. The blood was normal.

The mass in the throat was diagnosed as carcinoma by several competent observers (fig. 2, *12*). Unfortunately, no specimen was taken for biopsy, but the gland from the neck was removed and the diagnosis of squamous cell carcinoma was made. The patient was transferred to the St. Louis City Hospital, where, with the help of Dr. L. R. Sante, on November 15 and again on November 17, high voltage roentgen therapy was applied to the neck, as follows:

	Kilovolts	Distance, Cm.	Milliamperes	Filtration, Mm.	Time, Min.
11/15/30	200	50	20	1 copper, 1 aluminum	15
11/17/30	200	50	20	1 copper, 1 aluminum	15

On November 18, five gold seeds of 1 millicurie each, making a total of 726 millicurie hours, were implanted into the tumor by Dr. H. M. Janse and Dr. Dante.

As early as December 18 some regression in the size of the mass was noted, and by January 22, 1931, the condition appeared much improved. On February 26 there was marked regression in the size of the mass, and the voice was good. A second gland was removed on April 29. On June 24 there was no evidence of disease. On October 5 the larynx appeared normal in every way. On Jan. 2, 1933, except for a slight shortening of the right glosso-epiglottic fold, no abnormality of any kind or other evidence of disease could be seen in the larynx. Later in 1933, and again in May 1934, there were no signs of disease in the larynx or the neck, and the patient was working every day. On March 6, 1935, he was found to be entirely normal.

EXPERIMENTAL STUDY

In consideration of these and other cases, it appears that the lethal dose of gamma rays for cancer cells may be below that for cartilage cells and that the likelihood of injury to cartilage from the treatment may not be so great as some suppose. To test this hypothesis experimentally, similar seeds were implanted beneath the epithelium and close to the laryngeal cartilage of dogs, and inert seeds were likewise implanted in controls, as indicated in the table.

Some of the results are shown in the illustrations. The photomicrographs are arranged so that the direction of irradiation is from left to right, except in figure 2, *9*, showing a section of cartilage to the left of which an inert seed was implanted as a control measure and in figure 2, *11* and *12*, showing tissue obtained in human cases in which

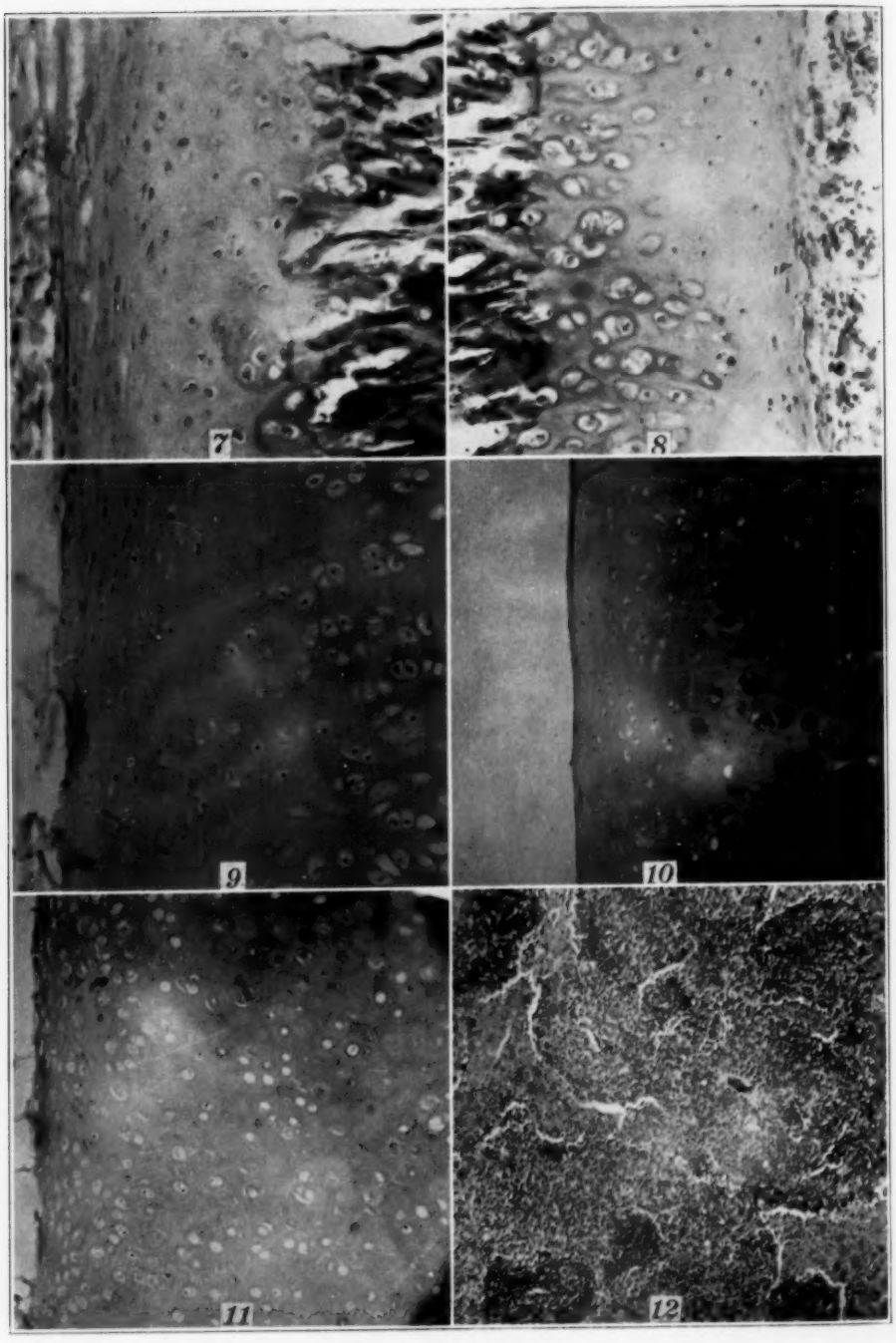

Figure 2

EXPLANATION OF FIGURE 2

Fig. 2.—Sections of laryngeal cartilage from dogs and from human subjects. Section *1* is from the same block of tissue as the section shown in figure 1, *6*. The perichondrium is on the left; the cartilage in the middle and the calcifying cartilage, on the right. In this case comparison with the other side of the cartilage (section *8*) remote from the seed does not reveal any marked differences. Section *8* is from the same block of tissue as section *7*. The surface of the cartilage is distant from the seed. Section *9* is from dog 8, a control, in which inert seeds were planted. This seed was 0.4 mm. from the cartilage. Section *10* is from J. B. (case 3), showing perichondrium and cartilage. The cells are injured, and the intercellular substance is more acidophilic. Section *11* is from J. O. (case 2) and shows degeneration of cartilage. Section *12* is from J. D. (case 4) and shows tumor tissue.

the direction was not accurately determined when the tissues were removed.

The observations were made on material fixed in Zenker's solution and stained with hematoxylin and eosin. In estimating the results of the experiments, attention was paid chiefly to sections which showed the location of the seed with reference to nearby cartilage. Some of the seeds were cut in making the sections, while others dropped out, in which case the position they occupied could readily be identified by the hole in the section and by the reaction of the tissue about it. Because of the unavoidable shrinkage of the tissue, the distances between the seed and the cartilage, given in the description of the figures, are probably about 12 per cent less than the actual distance which obtained in vivo.

The tissue reaction immediately about the active seeds exhibited features which were common to all as well as others which varied in

Data on Implantation of Gold Radon Seeds

Dog	Potency, Millicuries	Number Planted	Date Planted	Date Removed	Millicurie Hours
1	0.5	1	9/27/32	12/ 1/32	66.5
3	0.5	1	12/ 6/32	1/23/33	66.5
4	0.5	6	3/16/33	4/27/33	66.5
5	0.5	7	3/16/33	5/ 1/33	66.5
6	0.5	7	3/16/33	5/ 1/33	66.5
7 left	1.0	2	1/20/34	3/ 4/34	133.0
7 right	1.5	2	1/20/34	3/ 4/34	199.5
8 control	Inert	5	1/22/34	3/ 4/34	

individual cases. In the first category we list accumulation of fluid and coagulative necrosis characterized by the strong affinity of the material for acid dyes like eosin and extending a variable distance from the seed, depending on the nature of the tissue and the opportunity for spread of fluids. In loose connective tissue the effect is manifested a considerable distance away, say 0.5 mm., while cartilage seems to act as a restraining wall, limiting the area of necrosis to a layer of tissue as little as 0.1 mm. thick intervening between it and the seed. Striated muscle, when present near the seed, always behaved in the same way. It is convenient to recognize three zones, seen to the best advantage in muscles cut transversely: 1. Next to the seed and sometimes at a distance of 0.1 mm. from it all nuclei were destroyed, even those of fibroblasts. Cohnheim's areas were merged together, and the muscle substance was changed into an optically homogeneous, highly acidophilic mass. It is interesting that the diameter of these necrotic muscle fibers was not much reduced. They were killed quickly. 2. A little farther from the seed, however, the muscle fibers were distinctly but unevenly atrophied, some more and some less so. Cohnheim's areas

were indistinct, but their nuclei were prominent, of irregular shape and often situated in the central parts of the fibers. The appearances suggest that here the alterations were brought about more slowly and represent a response on the part of living cells. 3. Still farther removed from the seed was a zone of transition to normal muscle. The fibers were larger and striated, and their nuclei were peripherally placed.

Among the special changes we noted a variable neutrophilic response. In some instances so many of the neutrophils were grouped about the seed as to indicate an infective process. In no case were they absent, but some of them looked as if they had invaded the necrotic tissue after the primary injury, which it seems unlikely that they could have survived. Macrophages were sometimes present in abundance, though their distribution was rather irregular. In their investigation of radon seeds inserted into the liver, Rogers and Higgins [12] found that the most marked reaction was an enormous increase in these cells, which they called "histiocytes," and that this occurred in the first twelve hours. None of our sections of tissue was removed so early, and we were, of course, not dealing with a tissue potentially so rich in cells of this kind. In our specimens we often found an intense infiltration with macrophages on one side of the seed and not on the other. They were more numerous in the second zone of injured muscle. With them occurred a few lymphocytes and many plasma cells. The presence of the latter suggests that the infiltration was not very fresh, that is to say, a matter of a few hours, for the local metamorphosis of lymphocytes to plasma cells takes time, though exactly how long unfortunately is not known. We believe that the macrophages are called in to remove necrotic particulate material. Some were engorged with an orange-yellow substance and others with cytoplasmic and nuclear débris.

The effect of radiation on cartilage was much less noticeable than that on the muscle. Indeed it appeared to be so slight that we experienced great difficulty in characterizing and measuring it by the crude histologic technic employed in this preliminary study. We were unable to detect any consistent difference in cartilages exposed 66.5, 133 and 199.5 millicurie hours (table), but it was not feasible to measure the precise distances of the seeds from the cartilage with the heavier dosages. Unhappily, also, control cartilages from normal dogs were not sectioned in the same plane as the irradiated cartilages and then mounted side by side and stained with the same stains, thus permitting a detailed comparison. Consequently, we attempted to discover some difference between the side of the cartilage near the seed and that remote from it comparable to the conspicuous difference in the near and far parts of a band of muscle similarly situated.

12. Rogers, J. C. T., and Higgins, G. M.: The Effect of Radon Implants on the Cytology of the Liver in the Albino Rat, Radiology **22**:93, 1934.

No distinction is visible in figure 1, *1*, but the cartilage was extremely narrow where the section was made. Figure 1, *2* and *3*, shows the near and remote sides of a much wider piece. Here the side most directly exposed to the radiation (fig. 1, *2*) differs from the other side in reduction of the number of nuclei of the perichondrium, swelling of the intercellular substance as if water had been absorbed and an increase in the affinity for eosin, indicating perhaps a swing of the reaction toward the basic side. Obviously this decrease in nuclei does not mean loss of cells by degeneration, for terminal stages in nuclear disintegration were not seen. The cells with their nuclei were merely spread over a larger area by the edema. The fact that no phagocytic cells were seen in the perichondrium or cartilage in this or in any of the other preparations indicates that there is no dead particulate material which must be removed, as in the case of the killed muscle cells.

Comparison of *4* and *5* of figure *2* again reveals more marked acidophilia of cartilage near the seed as contrasted with that farther away. But *7* and *8* of figure 2 illustrate near and remote surfaces of another section of cartilage between which we observed no definite difference. The surface closest to the seed (fig. 2, *7*) was not more acidophilic than the other surface (fig. 2, *7*). Moreover, examination of normal cartilage sometimes shows that the cells and intercellular substance near one surface are more acidophilic than those of the opposite surface. We do not know why, but one thinks first of a possible difference in the quality of the blood supply. Though this difference in staining points unmistakably to a difference in chemical constitution, perhaps to alterations in the amount of chondroitin-sulphuric acid, we do not wish to emphasize it because of our inability to relate it definitely to the irradiation.

Figure 1, *6*, is instructive. The section through the cartilage was sufficiently narrow to be included in a print of the size presented. The surface most directly exposed to the seed (i. e., the left) has not the same appearance as the remote one. The cells are more pressed together, and the nuclei are more flattened. But there is no evidence of accumulation of fluid in the intercellular substance as represented in figure 1, *2*, also from the side of cartilage near the source of radiation. Evidently the edema, like the acidophilia, is present in some cases and absent in others, despite the fact that the tissue reaction about the seed was the same in both. This leaves us in doubt as to the responsible factor or factors.

Another alteration is illustrated in figure 1, *6*, namely, the deposition of material staining deep blue with hematoxylin in the middle of the cartilage due probably to beginning calcification. It is approximately equidistant from the near and the remote side with reference to the seed. The two much thicker sections of cartilage shown in figure 1,

2 and *3*, and figure 2, *7* and *8*, respectively, exhibit somewhat ragged, deeply stained internal borders which did not cut evenly and smoothly when the sections were made. This, likewise, is indicative of beginning calcification. It is our impression that infiltration with calcium salts is more marked in the irradiated cartilage than in the control specimens. Figure 2, *9*, showing a cartilaginous surface next an inert seed, does not show any signs of calcification. It may be compared with the irradiated cartilage immediately above in figure 2, *7*. Unfortunately, however, we did not make serial sections. There is a possibility that the sections were not similarly oriented. That depicted in figure 2, *7*, may have passed directly into the cartilage perpendicular with the surface, while the one shown in figure 2, *8*, may have almost skimmed the surface passing in at an acute angle and consequently containing only cells situated near the surface, which would be less prone to calcification.

These experiments, which we hope to extend, demonstrate clearly that seeds shown to be active by the tissue reaction about them do not bring about noticeable alterations such as necrosis and perichondritis in the cartilage at the distances specified in the description of the figures and at the times after implantation given in the table. In order to say with assurance exactly how the cartilage is influenced by radiation, it will be necessary: (1) to experiment with dogs of a single litter and therefore more directly comparable; (2) to identify each seed and the cartilage to which it is applied by careful dissection before removal; (3) to measure in the dissection the actual distance between the seed and the cartilage and also between the seeds themselves so that accurate allowance may be made for summation of radiation when they are close together; (4) to arrange the blocks of tissue for the preparation of sections so that there will be no question about the orientation, and (5) to employ more refined cytologic technic, including those of Policard, Péhu and Boucomont,[13] calculated to reveal even slight changes in the cells, the fibers and the ground substance.

SUMMARY

Regression of endobronchial carcinoma without clinical signs of loss of cartilage has been described in the reports of the cases. In J. D., who recovered, there is every reason to suppose that the cartilage remained more or less normal. The change in J. B. (fig. 2, *10*) was not severe, but we cannot predict what the ultimate consequences would have been. But in another case (J. O., fig. 2, *11*) the cartilage cells in tissue removed by bronchoscopy post mortem were unquestionably destroyed. In this instance suppuration (empyema) must be kept in mind as a

13. Policard, A.; Péhu, M., and Boucomont, J.: Recherches histopathologiques sur le rachitisme dans la première enfance: I. Le tissue chondroide, Bull. d'histol. appliq. à la physiol. **9**:73, 1932.

possible factor—cell destruction. While it is not our purpose to champion any particular form of treatment, we have come to believe that the combination of gold seeds planted directly into the tumor plus high voltage roentgen irradiation probably is more efficacious than roentgen irradiation alone. When complete removal is possible, we favor this as the method of choice for the treatment of malignant disease, but when this is not possible one may reasonably expect help from obstructive symptoms by the use of gold seeds. It is possible that with some improvements and more experience we may come to the point where we shall be able to treat successfully with radiation those persons who refuse surgical intervention for relief of malignant disease of the larynx.

The experiments demonstrate that active gold seeds may be implanted near cartilage in the dog's larynx without producing consistent and easily identifiable microscopic cartilaginous changes in the periods of time specified. Yet it would be unsafe to say that no injury is caused. Even if further and better controlled histologic studies fail to reveal alterations, the possibility will remain that a chemical injury may be sustained which may manifest itself morphologically and functionally at a considerably later date. Cartilage is one of the least cellular tissues in the body. It contains a large amount of inanimate intercellular material. On general principles one would not expect it to be very susceptible to radiation. The possibility remains, however, that on account of its avascularity and comparatively slow exchange of fluid slight modifications in it are more persistent and tend with repetition to summation in the course of time.

ALLERGY AND ITS RELATIONSHIP TO SINUSITIS AND ALLIED NASAL CONDITIONS

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It has been the experience of every physician specializing in the treatment of abnormal conditions of the nose and throat to wonder how next to deal with the cases of those patients who complain of alternating stenosis of the nose, sneezing and watery discharge. It seems that no matter what routine procedure is used, no permanent result is attained. Shrinkage of the nasal tissues, cautery and trimming of the turbinates give only temporary relief. In other words, as long as the patient continues to come for treatment the partial relief lasts, but as soon as the patient remains away for a short time the condition returns in all its fury. Many patients complain of headache and of a heavy feeling in the head, and roentgen examination reveals a thickening of the linings of the nasal sinuses. Intranasal examination almost always reveals pale, boggy, swollen mucous membrane, and since the lining of the sinuses is continuous with the mucous membrane which lines the nose it is fair to believe that the condition affecting the mucous membrane covering the turbinates also affects the mucous membrane lining the nasal sinuses. Hence the roentgen report of thickened membranes.

Formerly I sent all patients with a condition of this type to an allergist for skin testing, and the report always came back "negative to all tests." However, I was finally convinced that all such conditions have an allergic background; so about three years ago I started to do my own skin testing, recalling all the patients previously tested by others who were reported to give negative reactions. The results were so startling that I made skin testing a routine part of the examination in every case in which there was a history of sneezing, profuse watery discharge, hay fever and asthma, in those cases in which there was complaint of obstruction to nasal breathing and in those in which on intranasal examination pale, boggy, swollen mucous membrane was found.

Skin testing has been done for years, yet the results in the treatment of abnormal conditions in the nose and throat have not been satisfactory. Evidently something has been wrong in the way the condition was approached, in the way in which the tests were applied or in the reading of the completed tests. On investigation it was found that, in the first place, in most cases not enough tests were made; also little attention

was paid to the testing of foods in connection with abnormal conditions in the nose and throat; finally, the manner in which the tests were made by the average person doing the testing was very poor, for the arm was badly traumatized in the scratching, making the patient, especially if a child, afraid of the test and masking the true readings by the trauma of the skin.

I changed the common method of making the tests on the body. The usual method is the scratch test, in which a scratch is made on the arm, then a drop of decinormal sodium hydroxide is placed over the scratch and some of the protein is dissolved in the solution over the scratch. A different scratch is made for each protein tested. After from twenty to thirty minutes the reactions are read. Most persons who do the scratch test look for wheals in order to call a reaction positive. One must realize if one intends to make a large number of tests on a patient, especially on a child, that one must first have a method of testing that will not only give accurate results but will give the patient a minimum of pain and discomfort.

In conjunction with Dr. J. R. Taylor I devised what is known as the pressure puncture test. I believe that this method of testing is superior to the ordinary scratch or intradermal method because each opening made in the stratum corneum is of a standard size and is made with a minimum of trauma, thus allowing a close reading of the erythema caused by the protein. The test is made with the Taylor protein knife. The point is gently pushed through the stratum corneum, care being taken that blood is not drawn. Then a drop of Ringer's solution is placed over the mark of the blade and a speck equal in size to that of an average pinhead of the protein of the food to be tested is dissolved in the drop of Ringer's solution. Just as accurate a result is had by using a minimum amount of the protein as by using a large amount, for the latter results only in a waste of material. Ringer's solution is used instead of the generally used decinormal sodium hydroxide, for it does not cause a reaction, while a reaction often occurs with sodium hydroxide, even before the protein is added, when used on a very sensitive skin. After the test substance is placed on the arm, about twenty or thirty minutes are allowed to elapse. Then the arm is cleansed with Ringer's solution, and after about ten minutes the reading is made.

Experience has shown that if good results are to be had the reactions must be read closely, and I am convinced that this is the reason for the excellent results that I have obtained. The picture-book wheal, which is had when testing for sensitivity to pollens and grasses and which most persons look for in doing the testing, does not usually occur; instead, any test that causes an area of erythema that is larger than the

control is called positive. One must bear in mind that the patient is seldom as acutely sensitive to foods as he is to epidermal proteins and pollens. The patient partakes of foods in quantities ranging from ounces to half pounds, while it is agreed that a very small fraction of a milligram of epidermal protein or pollen may produce symptoms. I admit that in reading as closely as I do some questionable positive reactions are read and that therefore some foods that do the patient no harm are excluded from the diet, but the management is much easier and the patient is relieved of his difficulties much sooner if all foods causing a positive or questionably positive reaction are eliminated from the diet. Then when the patient is free from symptoms, the foods giving a questionably positive reaction can be added to the diet first. Occasionally a patient is tested who shows the picture-book wheal to every protein to which there is a positive reaction, but this is rare. To avoid missing any delayed reactions, the patient is examined twenty-four hours after the test is made.

As previously stated, it is essential that a test be made for all foods and excitants with which the patient may come in contact. The patient must be tested completely or failure is sure to result. Ten, twenty or thirty tests are not sufficient and result only in wasting the patient's and the physician's time.

Contrary to general belief, in a series of over 175 cases I have found that foods are the predominating factor in the causation of abnormal conditions in the nose and throat and that one must not stop with tests for reactions to inhalants, excitants, grasses and pollens. It seems hard to believe that foods play such an important part in cases of allergy in which the patient shows nasal manifestations, yet in my series of cases practically all the patients were relieved of their symptoms by eliminating from the diet the foods giving a positive reaction.

In this series of cases I dealt with patients who complained of snuffles, watery discharge from the nose and alternating stenosis of the nose, hay fever, sneezing and asthma. The results have been most satisfactory. The nasal symptoms cleared up almost immediately after the diet was started. Most patients reported, after being on the diet a certain length of time, that they could pin the responsibility for the condition down to a certain group of foods that I had excluded from the diet because of a positive reaction, thus allowing the replacing in the diet of many of the foods that previously caused a positive reaction but which later could be added to the diet without causing trouble. The final small group of foods always causes a recurrence of the previous symptoms when eaten. In other words, I can make the patient free from symptoms and then cause a recurrence of nasal symptoms by feeding him one or more of the offending foods. As long as the patient includes

these offenders in the diet he has nasal symptoms, but as soon as the offending foods are removed from the diet he again becomes symptom-free.

In those cases in which the turbinates are large, swollen and boggy the nose is examined after the patient has been on the diet about six months. Invariably intranasal examination reveals the turgescence and swelling to be gone and the nasal passages to be free and clear. This holds true also in those cases in which the mucous membrane droops down, covering the turbinates. As to how the patient has felt in the interim, I have only his word and if he is satisfied that the nasal breathing is clear, that the nose has stopped running and that he is completely relieved, then I as a physician must be satisfied, for in the last analysis the patient must be the judge. In cases of this type in order to hasten the reduction of the swelling of the nasal tissues, a few treatments are given consisting of shrinking the nasal tissues with a weak solution of cocaine and then with a silver preparation.

An interesting feature observed in the study of this group of cases was that, whereas one would expect the patient to lose weight on a diet from which in some cases many of the so-called weight-producing foods are eliminated, in the majority of cases the opposite was true. Substantial increases in weight were shown, especially in children; it was gratifying to the mothers to have their children whose weight was stationary for long periods of time start to gain weight.

The following tabulation gives a summary of the complications in the cases of 100 patients with hyperesthetic rhinitis treated by me in my office over a period of two years. Forty-two per cent were male and 58 per cent female.

Number of Patients	Complications
25	Hay fever
14	Asthma
2	Hives
4	Eczema
2	Canker

Satisfactory results were had in 93 patients; of the 7 in whom the treatment failed, only 2 cooperated by adhering to the diet, the remaining 5 paying no attention to the diet. Thus, I can say that in the entire group there was failure in only 2 per cent.

During the past year, in connection with the otolaryngologic, pediatric and food clinics of the Boston Dispensary, Dr. S. Dushan and I carried out a study of the cases of 75 children under the age of 12 years. These children complained for the most part of sinusitis, colds, headache and asthma. Among them were a few with eczema who were referred for testing. Many of the children had been coming to the hospital for a number of years and had been given various forms of treatment; a few scratch tests had been made, from which very limited

results were obtained. When the patients were tested and placed on a diet from which the foods that caused a positive reaction had been eliminated the results were very satisfactory. For the first time in their lives many of the patients were free from symptoms, suffering no further nasal trouble or asthma.

The children in this group were first given a thorough physical examination in the pediatric department, and a complete family history was taken. I then made a complete examination of the nose and throat in the otolaryngologic clinic. If we were satisfied that the case had an allergic background, the child was tested for all the common inhalants, excitants, grasses, pollens and foods with which he might come in contact. After the tests were completed, a list of the proteins causing a positive or negative reaction was made out and sent with the patient to the food clinic, where, under the direction of Miss Frances Stern and Miss Catherine Hazen, a proper diet was planned. The patient was closely observed for a time to make sure that the instructions were followed exactly. This group of patients had to be watched carefully, and full cooperation of the parents of the patients was demanded. When one remembers the difficulties in dealing with a group of patients in a clinic who are handicapped by a limited income, one can realize the excellent piece of work that was done.

There were 35 patients, 57 per cent of whom were male and 43 per cent, female. There was a familial tendency to the condition in 59 per cent. The following tabulation shows a partial analysis of the conditions in some of the cases:

		Cases
Cold and sinusitis		23
Uncomplicated cases	7	
Sinusitis and hay fever	1	
Sinusitis and asthma	5	
Sinusitis and eczema	4	
Sinusitis, hay fever and asthma	1	
Sinusitis, eczema and asthma	3	
Sinusitis and gastro-intestinal disturbance	1	
Sinusitis and hives	1	
Asthma alone		2
Hay fever alone		3
Eczema alone		4
Asthma and eczema		2
Asthma and hay fever		1

The results were as follows:

	Percentage
Entire relief while on diet	49
Much relief while on diet	34
Some relief while on diet	6
Failures	11

Thus there were good results in 83 per cent of the cases and some relief in 6 per cent. It must be borne in mind that in dealing with a group of this kind the income plays an important part. The average weekly income of the families of the patients in this group was $16.30

for a family of 5.1 persons, which one must admit is small when buying the necessities of life.

The causes of failure in the 11 per cent of the cases in which there was no relief are as follows: (1) failure to follow diet; (2) failure to return; (3) lack of income which the clinic could not supplement; (4) no cooperation when there was no relief, and (5) sensitivity to excitants that it was not possible to remove under the circumstances.

Important points in the treatment were: 1. After the patient is free from symptoms too many foods should not be added to the diet at one time. 2. It is easier for the child to assume the responsibilities than the mother, who is not always as cooperative as the child. 3. Frequent visits for assistance and encouragement are helpful. 4. A strict diet should be maintained for the first three months at least.

Taking all the circumstances into consideration, I think that the results in the children were very good; they far exceeded our expectations. The group was followed over a long enough time so that no question would arise as to the occurrence of symptoms at some time of the year when the patient was not observed.

It must be remembered that the task is not completed when the tests are made and the patient is told to eliminate this, that or the other thing from the diet. Especially is this true in children, for they require vitamins, minerals and calories to meet body requirements, and other foods must be substituted for those removed. After the patient is tested he is referred to a dietitian who works in conjunction with me and who makes the proper substitutions of food. In this way, it is insured that the patient has a full, well balanced diet that is not deficient in any of the things vitally necessary to good health. When a certain food that makes up the principal source of a mineral, such as milk which supplies the bulk of the body's need of calcium, is eliminated, the mineral is supplied in chemical form. For example, to supply calcium there is di-calcium sodium phosphate which is marketed in pleasant-tasting wafers or dulcets. Another important thing to teach the patient is the presence of some of the substances causing a positive reaction hidden in foods. For example, eggs are present in ice cream and pastries. The three main groups with regard to which care must be taken in this respect are eggs, milk and wheat.

In the case of those patients who suffer mainly from hay fever, it is not sufficient to test for pollens, trees and grasses and then give injections against these excitants. In every case in which there is a positive reaction to pollens, trees, grasses and epidermal protein one will gain a positive reaction to certain foods. The results from the injection of extracts of pollen alone have been very poor, but the injection of extracts of pollen plus a diet free from foods which cause a positive reaction gives excellent results.

During the past hay fever season, I omitted the injection of pollen extract in 10 cases and placed the patient on a diet free from proteins which caused a positive reaction. The results were very good. As long as the patient, including myself, as I suffer from hay fever, adheres strictly to the diet, there is no hay fever, but let the patient deviate from the diet and immediately the sneezing and running from the nose commence and continue until the offending protein is out of the system. I have not yet had enough cases to enable me to draw a definite conclusion in this matter, but it will be interesting to continue the study to see whether or not the patients are basically sensitive to food and secondarily sensitive to pollen, inhalants and grass.

CONCLUSIONS AND SUMMARY

1. Foods play an important part in the causation of certain symptoms and conditions in the nose and throat.

2. Foods play an important part in the causation of symptoms during the hay fever season in sufferers from hay fever.

3. Proper skin testing and reading of the reactions make possible detection of the offending foods, excitants, inhalants, trees, grasses and pollens.

4. Removal from the diet of the foods which cause a positive reaction, in addition to removal of the offending excitants and inhalants, alleviates the symptoms.

5. Removal from the diet of foods causing a positive reaction, in addition to inoculation with extracts of pollens which cause a positive reaction, materially lessens the number of attacks of hay fever.

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CHRONIC SUPPURATIVE SINUSITIS

A POINT OF VIEW AS TO TREATMENT

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So much has been written in recent years about the necessity of radical external operations and the meticulous removal of every vestige of lining mucous membrane in chronic suppurative pansinusitis that one wonders whether the surgical axiom of adapting an operation to the pathologic condition encountered rather than adapting the patient to a certain operative technic still holds true.

The technical approach to the surgical problem involved in the successful management of chronic suppurative sinusitis may in a large measure be dependent on the capability of the surgeon.

It would be entirely contrary to my clinical and surgical experience to concede that it is necessary to remove every vestige of lining mucous membrane in every case of chronic suppuration of a paranasal sinus. It is frequently claimed that the membranes should be removed because bacteria are found in specimens of membrane that are examined. The validity of this claim may honestly be questioned. The thoracic surgeon does not remove the lung because bacteria are found in and on the pleura in empyema. In bacteremia one hopes only to remove the source of infection so as to permit the patient to combat sepsis more easily by natural processes. It is admitted that the kidney, gallbladder or appendix may need to be removed because of the presence of serious infection; by removal of the organ the lesion is removed and infection cannot recur. If the removal of all the mucous membranes from the sinuses is justified on the supposition that the disease is removed for all time and that recurrences of infection of the upper part of the respiratory tract will not take place, perhaps the procedure is logical. But will clinical experience bear out such a probability? There can be little doubt that the lining membrane should be conserved if at all possible. It has not been proved to the satisfaction of all observers that the membranes regenerate in a normal manner. Too many patients recovered following adequate surgical intervention before the removal of the mucous membrane was advocated as a principle to reconcile one to applying the principle to all cases.

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With the increase in knowledge of the etiology and pathology of hyperplastic or polypoid changes in the membranes in the nose and paranasal sinuses it would seem necessary to study the patient thoroughly from the standpoint of allergy and endocrine disturbances before instituting a radical surgical procedure. When suppuration and hyperplasia coexist, deciding on what is best to do may be difficult. It is questionable whether operative intervention should be decided on at once. Much time may be gained and better results may be had if the patient is studied from the standpoint of allergy and endocrine disturbances. If these causative factors of polypoid and hyperplastic changes can be controlled the suppurative process may respond readily to adequate surgical intervention. If the hyperplasia does not respond to a well directed regimen the most extensive surgical measures may be employed. Even such measures may fail so far as the hyperplasia is concerned. Seldom, if ever, in this type of case are symptoms and signs present which cause the case to be classified as an emergency.

Adequate treatment should include a period of observation in cases in which the clinical picture of infection of individual sinuses is not sufficiently clear to permit the surgeon to determine which, or how many, of the sinuses are involved. The presence of pus in certain positions in the nose at the time of the first inspection is no indication of its origin. Because of blowing of the nose, snuffing and so forth, the pus may be situated in any position. If the pus is removed and some time is permitted to elapse, another inspection may more accurately reveal the origin of the pus, particularly if suction is effective. Several examinations on separate days may be necessary to determine satisfactorily the extent of involvement. During this period one is subconsciously determining what measures will be necessary to control the condition adequately. For example, the effect of the presence of pus on the mucous membranes of the middle meatus suggests in many cases that the ethmoid cells or the frontal sinus is involved, whereas the changes may be caused by pus from the antrum. Draining the antrum through the inferior meatus as a first step in the care of the patient may cause such a marked change in the appearance of the membranes of the middle meatus in a very few days that the tendency to explore the ethmoid cells and frontal sinus may be obviated.

It seems self-evident that what may be considered a radical procedure for one patient may be considered conservative for another. It does not seem necessary to use the terms "conservative" and "radical" in the treatment of sinusitis. It seems more logical to individualize the treatment for the particular case and to institute "rational" treatment.

Much of the postoperative care of the patient may be accomplished at the time of the operation on the paranasal sinuses. This implied thoroughness does not necessarily mean the removal of all of the mucous membrane.

Meddlesome postoperative treatment may, and in fact does, prolong the convalescence. After apparent recovery it is best to check over the intranasal condition occasionally for a period of months. My experience with operations on the frontal sinus particularly has made me feel the need for an occasional inspection of the nose for some time.

A patient should be required to remain under observation for a sufficient length of time to receive the maximal amount of benefit and to permit the surgeon to be satisfied with the result. "Changing horses in midstream" is unsatisfactory without mutual consent.

In general, results are good although they may not be 100 per cent perfect from the standpoint of either the patient or the surgeon.

The following report of a case is illustrative:

REPORT OF A CASE

A married woman, aged 27, complained of an excessive amount of thick mucopurulent discharge from the nose and severe headaches over the frontal sinuses. Many emotional disturbances had developed as a result of the symptoms, which had been present for more than five years. She looked and acted like a patient who would be difficult to take care of. She had received much treatment and had had several nasal operations. Because she had been advised to undergo a bilateral external radical operation on the sinuses and because she was reluctant to have this type of operation performed she presented herself at the Mayo Clinic for consultation.

As home treatment she had frequently used nasal irrigation with a solution of salt and soda.

Examination disclosed that the nasopharyngeal membrane was covered with a thick, tenacious mucopurulent discharge which was difficult to remove. The right nostril was filled with a similar discharge. After removal of the secretions a large window into the antrum was discerned. The anterior portion of the inferior turbinate bone had been removed. Pus was seen between the middle turbinate bone and the nasal septum. A spur which was situated high on the septum interfered considerably with the introduction of instruments into the sphenoid sinus. The nasofrontal duct could be probed easily. The left nostril presented about the same picture except there was no spur on the septum. However, the opening into the sphenoid sinus could not be probed. Pus came from the nasofrontal duct on probing. It was decided to study the nose carefully before making suggestions as to treatment. The patient was asked to discontinue the nasal irrigations because the effect of water-logging was very apparent. During the first week of observation great improvement took place; the discharge lessened greatly, and it could be seen to come from the posterior ethmoid cells and sphenoid sinus on the right side and from the frontal sinus and ethmoid cells on the left side.

An intranasal operation was advised as the first procedure; this was to be followed, if necessary, by an external operation on the left frontal sinus. In order to reach the right sphenoid sinus it was necessary to remove the right middle turbinate bone. The anterior wall of the sphenoid sinus was removed. The sphenoid sinus contained much pus; there was thickened mucous membrane in several pockets. One pocket was found to pass across the median line to the left side. This appeared to be the only sinus involved on the right side, but the

posterior ethmoid cells were explored. The left nasofrontal duct was enlarged and the anterior ethmoid cells in the agger nasi were exenterated. The middle turbinate bone was removed. There was no natural opening in the left sphenoid sinus, so the anterior wall was removed and the large pocket was found.

So far as the nose was concerned the postoperative course was satisfactory. After a week the patient became so homesick that she begged to be permitted to go home for a few days. It seemed best to grant the request, for the condition obviously was satisfactory. She returned in two weeks greatly improved in general, and the nose was in excellent condition except for discharge from the left frontal sinus. She could not remain at this time for further surgical treatment. Two months later she returned and complained of pain over the left frontal sinus. The observations were as previously noted, and in addition the nasofrontal duct could not be probed and the roentgenologic examination disclosed that the shadow of the left frontal sinus was darker than that of the right. An external operation was advised and at this time urged. The bone around the nasofrontal duct was found to be much sclerosed, which verified the clinical impression that it would have been impossible to do an intranasal operation satisfactorily. The frontal sinus was cellular, and the removed thickened mucous membrane contained pus. The fronto-ethmoid cells extended flatly out over the orbit. These were uncovered, and all the ethmoid cells were obliterated to the sphenoid. This made a very satisfactory exposure of the field.

There was swelling of the wound after the operation, but this subsided after a few days. The convalescence was rapid, and the patient felt so relieved that she insisted on going home on the sixth day after the operation. She left, of course, against my advice.

COMMENT

The deleterious effect of the continuous use of aqueous nasal irrigations seems apparent. I have previously stated that the only apparent reason for the use of such a method of treatment was its time-honored tradition. As my experience increases I feel more convinced of it. Certain patients apparently are not effected in the same manner, but the majority do better if the method is not used.

In the case reported here a great decrease in the amount of secretion took place in a few days when the irrigations were discontinued. The excessive amount of tenacious mucopurulent discharge resulted from water-logging of the membranes. The discharge resulting from the infection was of a very different character.

It does not seem necessary to utilize an external method when only the posterior ethmoid cells and sphenoid sinuses are involved. In the case reported here, because the natural opening of the left sphenoid sinus was not made out and because it was known that the right sphenoid sinus extended past the median line, the anterior wall of the sphenoid bone on the left side was boldly removed. The observations were as noted in the surgical description. With suitable instruments the opening may be made as large as necessary.

It may be safer to perform an external, rather than an intranasal, operation for the relief of a condition in the frontal sinus when there is

evidence of sclerosis of the bone which is the result of long continued infection. As a matter of fact, the external operation for involvement of the frontal sinus is not particularly difficult from a technical standpoint.

Failure of the patient to cooperate with the surgeon in the after-care is a difficult problem to overcome. However, it has been observed that if the operation is carefully carried out little else need be done other than to remove mechanically the products of repair. Patients who are forced to leave before the convalescence has been completed frequently return for observation in excellent condition.

PATHWAYS OF REFLEX PAIN IN VIDIAN NEURALGIA

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Because Garber [1] doubts that sympathetic fibers ever act as pain conductors he has criticized my [2] use of the sympathetic nervous system to explain the subjective pain in the neck and shoulders in patients with vidian neuralgia. In addition he has stated that "the vidian nerve should not be considered as having anything to do with the neuralgic pains of Sluder's syndrome and a better explanation is afforded by regarding the maxillary nerve as the one involved."

Before answering his criticism and theory that the starting point of the reflex neuralgia about the head is the maxillary nerve and not the vidian nerve, it may be well to review briefly the syndrome which Sluder [3] classed as Meckel's ganglion neuralgia and which I have called vidian neuralgia.[4]

In this form of neuralgia a sharp deep pain starts in the root of the nose, radiates in, about and behind the eye, over the temple to the ear and the mastoid process to the back of the head and the neck and in severe cases passes into the shoulder and the arm; it continues steadily for from hours to days, is not associated with any sensory disturbances or caused by external stimulation of the face and is completely relieved in a few minutes by the injection of a few drops of 10 per cent solution of cocaine into the sphenoid sinus of the affected side.

Anatomic studies show that there may be an exceedingly close relation between the vidian nerve and the mucosa of the floor of the sphenoid sinus, and by clinical examination with opaque oil and the roentgen rays and on surgical intervention there is always found in cases of vidian neuralgia an extensive pneumatization of the sphenoid sinus with recesses on one or both sides of the vidian canal which may result in the projection of the thin bony wall of the vidian canal as a rounded ridge above the floor of the sphenoid sinus, surrounded frequently on more than one-half its circumference by sphenoid sinus mucosa. These

1. Garber, H.: Sinusitis and Neuralgia, Arch. Otolaryng. **18**:339 (Sept.) 1933.

2. Vail, H. H.: Vidian Neuralgia, Ann. Otol., Rhin. & Laryng. **41**:837 (Sept.) 1932.

3. Sluder, Greenfield: Etiology, Diagnosis, Prognosis and Treatment of Sphenopalatine Ganglion Neuralgia, J. A. M. A. **41**:1201 (Sept. 27) 1913.

4. Vail, H. H.: Vidian Neuralgia from Disease of the Sphenoidal Sinus, Arch. Surg. **18**:1247 (April) 1929.

pterygoid recesses favor the development of latent sphenoiditis which flares up from time to time without producing the usual symptoms associated with a cold in the nose. The diagnosis of vidian neuralgia depends, therefore, on the typical pain, its relief by the injection of cocaine into the sphenoid sinus and its recurrence with subsequent attacks of acute sphenoiditis.

In the discussion of the pathways of reflex pain in vidian neuralgia the following sensory structures must be considered: (1) the peripheral and central portions of the trigeminal nerve; (2) the cutaneous sensory branch of the facial nerve, or nerve of Rhinehart; (3) the peripheral and central portions of the glossopharyngeal and vagus nerves, and (4) the intermediate nerve, the geniculate ganglion with its peripheral branches and the great superficial petrosal, the geniculotympanic and the external petrosal nerve.

Garber has expressed the belief that the great superficial petrosal nerve is the cranial autonomic component of the sphenopalatine ganglion. I believe that Kuntz [5] has shown that the great superficial petrosal nerve contains not only preganglion fibers but sensory fibers, which have been traced through the geniculate ganglion along the intermediate nerve into the medulla.

Fenton and Larsell [6] have confirmed the sympathetic character of the sphenopalatine ganglion and have stated that the geniculate ganglion belongs to the craniospinal series of the sensory ganglions, that its cells are of the unipolar sensory type and that there are also fibers of the sensory type in the great superficial petrosal nerve.

Larsell and Burns [7] stated that the primitive palatine branch of the facial nerve which in the beginning contains only visceral sensory fibers becomes modified into a mixed nerve and that there is formed on it a sympathetic ganglion, the sphenopalatine, which is composed entirely of multipolar sympathetic ganglion cells and to which pass numerous fine myelinated fibers which have their origin in the bulb. These fibers together with those of the palatine branch of the seventh nerve constitute the great superficial petrosal nerve. They further stated that these sensory and palatine fibers are not interrupted in the sphenopalatine ganglion but pass through it and join with branches of the maxillary nerve to form the palatine and posterior nasal nerves. They quoted the work of Kuré and Sakurasawa, who found that the unmyelinated

5. Kuntz, Albert: The Development of the Sympathetic Nervous System in Man, J. Comp. Neurol. **32**:173, 1920.

6. Fenton, R. A., and Larsell, O.: Mechanism of Pain Transmission in Certain Types of Otalgia, Ann. Otol., Rhin. & Laryng. **27**:739 (Sept.) 1928.

7. Larsell, O., and Burns, E. M.: Some Aspects of Certain of the Cranial Nerves, Ann. Otol., Rhin. & Laryng. **40**:661 (Sept.) 1931.

fibers in both the vidian and the maxillary nerve completely disappear after extirpation of the superior cervical ganglion and hence are sympathetic or thoracolumbar autonomic fibers. The Japanese observers expressed the belief that the unmyelinated fibers come from the deep petrosal nerve, the internal carotid plexus and the superior cervical ganglion and pass through the sphenopalatine ganglion into the zygomatic temporal nerve. Other secreting fibers must come to the sphenopalatine ganglion through the maxillary roots since lacrimal secretion continues after section of the vidian nerve and the chorda tympani. Larsell and Burns further stated that the sensory fibers of the great superficial petrosal nerve have their ganglion cells in the geniculate ganglion, in which are also the ganglion cells of the sensory cutaneous branch of the facial nerve.

In addition to the sensory nerve supply there is an extensive network of cranial autonomic and sympathetic pathways and connections. This was well brought out by Kuntz, who stated that a sympathetic ganglion develops with each branch of the gasserian ganglion, i. e., the ciliary ganglion with the ophthalmic division, the sphenopalatine ganglion with the maxillary division and the otic ganglion with the mandibular division. He further stated that the primordium of the ciliary ganglion rises from the oculomotor and ophthalmic nerves and that of the sphenopalatine ganglion develops from cell migration along the great superficial petrosal nerve from the geniculate ganglion, while the primordium of the otic ganglion develops from an accumulation of cells at the extremity of the lesser superficial nerve. In later embryologic life more and more cells from the respective branches of the gasserian ganglion pass into these three ganglions.

Any pathway for reflex pain must utilize either the sensory or the sympathetic system or both. With regard to the ability of the cranial autonomic nervous system to transmit painful stimuli there are two opposing opinions. Langley [8] and the authors quoted by Garber have expressed the opinion that the cranial sympathetic nervous system contains only efferent fibers. On the other hand, there is a more modern belief, the result of clinical observation, that the cranial sympathetic nervous system may transmit painful stimuli.

Evidence that the sympathetic system has an afferent mechanism is found in the report of Nelson [9] on a large series of cases of neuralgia of the trigeminal ganglion after section of the sensory root. He stated

8. Langley, J. N.: The Autonomic Nerve System, Cambridge, England, W. Heffer & Sons, Ltd., 1921.

9. Nelson, Harry: The Part Played by the Sympathetic System as an Afferent Mechanism in the Region of the Trigeminus, Brain **55**:114, 1932.

that the sensitivity to extreme heat which was noticed by the patient after section of the sensory root of the gasserian ganglion was entirely lost when, in addition to section of the sensory root, a thoracic sympathectomy had been performed. He felt that the sympathetic and parasympathetic systems have afferent as well as efferent functions. The work of Davis and Pollock [10] and Davis [11] kept the question open.

Frazier,[12] in discussing the question of the sympathetic pathways in pain, stated that operations on the periarterial plexus and removal of the superior cervical ganglion do not give favorable results.

Garber, according to the opinion I can gather, was only partly correct in the statement that there are no afferent fibers in the sympathetic nerves. Unfortunately the term sympathetic nerves is apt to be used rather loosely.

According to Grinker,[13] there are no true sympathetic afferent nerves, but there are so-called visceral or somatic afferent nerves traveling by way of the sympathetic trunks which have their cell bodies in the posterior ganglion and enter the spinal cord by way of the posterior gray horn. The afferent course of these visceral afferent fibers over the sympathetic system and their synapse with the same or related horn cells explain the mechanism of referred pain from inflammation of a viscus.

Vogel [14] showed by serial study of microscopic sections that there is a definite anastomosis between the orbital branches of the sphenopalatine ganglion and the terminal branches of the ophthalmic division of the trigeminal nerve within the orbit. There is a communication between the tympanic branch of the glossopharyngeal nerve and the geniculotympanic branch of the geniculate ganglion. The exact relation between the sphenopalatine ganglion and the nasopalatine nerves from the maxillary division of the trigeminal nerve is not definitely known, although most authorities feel that these nerves pass around the ganglion, especially as microscopic examination of the ganglion reveals its type of cell to be entirely sympathetic.

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10. Davis, Loyal, and Pollock, Lewis J.: The Rôle of the Sympathetic Nervous System in the Production of Pain in the Head, Arch. Neurol. & Psychiat. **27**:282 (Feb.) 1932.

11. Davis, Loyal B.: The Surgical Treatment of Intractable Pain, J. A. M. A. **101**:1921 (Dec. 16) 1933.

12. Frazier, C. H.: The Problems in the Diagnosis and Treatment of Trigeminal Neuralgia, New York State J. Med. **31**:1137 (Sept. 15) 1931.

13. Grinker, H. H.: Neurology, Springfield, Ill., Charles C. Thomas, Publisher, 1934, p. 267.

14. Vogel, Klaus: Die Beziehungen des Ganglion sphenopalatinum zu den von ihm ausgehenden Nervenstämmen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**: 435 (March) 1930.

Sufficient experimental work has been done by Vogel,[15] Schatz,[16] Moulonguet and Collin [17] and Jung, Tagand and Chavanne [18] on the sphenopalatine ganglion to show that its removal has no effect on the nasal mucosa or its secretion and that no lasting anesthesia results. The extent of the anesthesia which follows its removal is about the same as that which follows the injection of a local anesthetic into the ganglion.

The argument advanced by Garber that the fact that "the vidian nerve does not act as a conductor of pain impulses can be proved clinically by the fact that blocking of the gasserian ganglion gives complete anesthesia over the area supplied jointly by the fifth and vidian nerve" does not take into consideration the important fact that the pain in vidian neuralgia is deep and not located on the surface of the skin or mucous membrane and that deep facial pressure and pressure pain in the face are undisturbed by resection of the fifth nerve.

Garber's statement that the maxillary nerve is in just as close proximity to the sphenoid sinus as the vidian nerve is contrary to the known anatomic facts. The maxillary nerve lies laterally to the sphenoid sinus, and rarely is it in relation with even the wall of the sphenoid sinus, much less projecting into the cavity of the latter. Furthermore the nerve passes into the sphenomaxillary fossa, where considerable loose areolar tissue separates it from the sphenoid sinus. The intimate relation between the vidian nerve and the sphenoid sinus can be demonstrated in about one third of the anatomic specimens. Garber's theory that it is unreasonable to suppose that the involvement of a small nerve can give rise to pains, either direct or referred, which are felt over a large area is not to the point. The vidian nerve is relatively not small, and severe irritation of it from inflammation in the sphenoid sinus can occur over a comparatively large extent of its course. From the extent of referred pain in appendicitis, angina pectoris, renal colic and ureteral stone, one might be led to believe that the nerve involved in these conditions is large, which is not the case.

Garber used the well known connection which the descending root of the trigeminal nucleus has with the facial glossopharyngeal and vagus nuclei and the upper part of the cervical portion of the cord to

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15. Vogel, Klaus: Histopathologische Befunde am Ganglion sphenopalatinum mit besondere Berücksichtigung der strophischen Rhinitiden, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:507, 1929.

16. Schatz, K. W.: Welchen Einfluss hat das Ganglion sphenopalatinum auf die Entstehung der Ozäna? Ztschr. f. Laryng., Rhin., Otol. **17**:312 (Jan.) 1929.

17. Moulonguet, André, and Collin, Pierre: Quelques expériences sur le rôle trophique du ganglion sphenopalatin, Ann. d. mal. de l'oreille, du larynx **48**:197, 1929.

18. Jung, L.; Tagand, R., and Chavanne, F.: Sur l'innervation excito-sécrétoire de la muquese nasale, Compt. rend. Soc. de biol. **95**:835, 1926.

explain the pain in the neck and shoulders in his theory of involvement of the maxillary nerve; yet in conditions in which there is every reason to believe that the maxillary nerve is definitely irritated, as in tic douloureux or trifacial neuralgia, there is no overflow of pain down the neck and shoulder. The pain is confined to the actual area supplied by the part of the trigeminal nerve affected. For the aforementioned reasons I feel that Garber's theory that the maxillary nerve is the one involved in Sluder's syndrome, or vidian neuralgia, cannot be seriously considered.

Fibers from the superior cervical sympathetic ganglion unite with the first four cervical nerves, the hypoglossal nerve and the ganglions of the glossopharyngeal and vagus nerves. The carotid nerve also arises from the superior cervical sympathetic ganglion and forms the carotid plexus from which arises the second component of the vidian nerve, the great deep petrosal nerve, which is believed to be entirely sympathetic.

The middle cervical sympathetic ganglion sends fibers to the fifth and sixth cervical nerves. It is believed that all the fibers from below the level of the middle cervical sympathetic ganglion are efferent. If this is the case, any stimulus reaching the superior cervical sympathetic ganglion would pass down the cord and would involve the cervical nerves, producing the reflex pain in the neck, shoulder and arm. Any stimulus passing down the sympathetic fibers could act on the blood vessels in such a way as to result in reflex pain.

As there are afferent sensory and efferent sympathetic fibers in the great superficial petrosal nerve, presumably these are present in all the branches of the geniculate ganglion. Through the anastomosis between the orbital branches of the sphenopalatine ganglion and the terminal fibers of the ophthalmic division in the orbit and the geniculotympanic branch from the geniculate ganglion and the tympanic nerve from the glossopharyngeal ganglion in the cavity of the middle ear, definite peripheral reflex arcs are established between the great superficial petrosal nerve and the ophthalmic and glossopharyngeal nerves.

The pathways of reflex pain in vidian neuralgia, I feel, are as follows: The irritation affecting the great superficial petrosal nerve passes to the sphenopalatine ganglion along the sympathetic efferent nerves through its orbital branches to the anastomosis which these have with the terminal branches of the ophthalmic division in the orbit; the sensation is perceived by means of the latter and recognized by the higher centers as pain in back of the eyeball, along the side of the nose and in the temple. Through the sensory fibers in the great superficial petrosal nerve the stimulus is carried back to the geniculate ganglion. From this ganglion it is carried through the sympathetic efferent nerves of the geniculotympanic branch to the tympanic plexus, where it is taken up by the glossopharyngeal nerve and carried to the higher centers as

pain in the ear. A severe stimulus or an overflow passes over the glossopharyngeal and vagus nerves or perhaps through the deep petrosal nerve to the superior cervical sympathetic ganglion and thence to the cervical sensory nerves and is perceived by the higher cortical centers as pain in the neck, shoulder and arm. A longer reflex arc may be from the geniculate ganglion through the intermediate nerve to the medulla and to the tractus solitarius, composed of descending fibers of the seventh, ninth and tenth nerves. This is apparently the same course as that for the taste fibers from the chorda tympani. As disturbances of taste are not complained of by patients with vidian neuralgia, probably this longer reflex arc is not used.

The cutaneous sensory branch of the facial nerve, or the nerve of Rhinehart, has been utilized by Fenton and Larsell [19] to explain reflex otalgia. This is a possible pathway, and though I believe that most of the evidence points to the participation of the glossopharyngeal nerve in vidian neuralgia in the production of reflex pain in the ear, the nerve of Rhinehart must serve as a pathway in other conditions, such as otalgia dentalis and neuralgia of the geniculate ganglion. That a sensory branch exists has been shown by Ramsay Hunt.[20]

The recent report of Reichert,[21] who sectioned the glossopharyngeal nerve intracranially for the relief of severe neuralgia in the ear, is most convincing. Reichert's case is so important that I believe it should be described here in some detail.

A woman 31 years old, a telephone operator, complained of severe pain in the ear and drawing and discomfort in the upper part of the face, cheek, forehead and occipital region on the left side. A few days after a cold she noticed a sharp stabbing pain deep in the external auditory canal which caused her to shriek and hold her ear. Relief was obtained for twelve days by the injection of procaine hydrochloride into the sphenopalatine ganglion. Later injections gave no relief. Besides the pains in the ear there were at times an itching of the anterior wall of the meatus, aching pains in the left side of the face, nose, eyeball and occipital area and sensitiveness in the mastoid region. The sinuses were operated on, teeth were extracted and procaine hydrochloride was again injected into the sphenopalatine ganglion, without gaining relief. Finally the paroxysms appeared every one to three minutes. The surgical method employed was the cerebellar approach of Dandy, with local anesthesia. When the bundle containing the

19. Fenton, R. A., and Larsell, O.: The Embryology and Neurohistology of Sphenopalatine Ganglion Connections: Contribution to the Study of Otalgia, Tr. Am. Otol. Soc. **18**:183, 1928.

20. Hunt, Ramsay J.: On Herpetic Inflammations of the Geniculate Ganglion: A New Syndrome and Its Complications, J. Nerv. & Ment. Dis. **34**:73 (Feb.) 1907.

21. Reichert, Frederick L.: Tympanic Plexus Neuralgia; True Tic Douloureux of the Ear or So-Called Geniculate Ganglion Neuralgia: Cure Effected by Intracranial Section of the Glossopharyngeal Nerve, J. A. M. A. **100**:1744 (June 3) 1933.

seventh and eighth cranial nerves was touched and moved the patient stated that she felt pain in the auditory canal but it was not the tic pain. The ninth nerve was then touched, which caused her to shriek and exclaim that this produced the tic pain. Four times the nerve was touched with the same result. The glossopharyngeal nerve was then cut, and four months later there was no suggestion of pain. Sensation was lost over the soft palate on the left side, the pharyngeal wall from 2 cm. within the eustachian tube to the tip of the epiglottis and the posterior third of the tongue, where taste was lost.

In conclusion, I feel that the general ideas as to the reflex pathways in vidian neuralgia advanced by me in 1932 are correct. Furthermore, there is not sufficient anatomic, experimental or clinical evidence to assign to the maxillary nerve, as Garber desires to do, the same rôle in vidian neuralgia as has been credited by me to the vidian nerve.

PRINCIPLES UNDERLYING CILIARY ACTIVITY IN THE RESPIRATORY TRACT

III. INDEPENDENCE OF TRACHEAL CILIA IN VIVO OF DRUG AND NEUROGENOUS STIMULI

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Certain previous investigations of the responses of cilia and ciliated epithelia to chemical substances and drugs have involved bringing the cilia into direct contact with the experimental fluid being considered. This procedure is applicable when the cilia to be studied are bathed in their normal habitat by sea water, fresh water or body fluids but can hardly be indicated as an altogether desirable method for the study of ciliated epithelium of the respiratory tract. Moreover, the majority of these studies on the cilia of the respiratory tract have been made on excised tissue, which treatment may lead to error when it is sought to interpret the results in terms of functional behavior in situ. Evidence of the validity of this criticism, at least in one instance, has already been demonstrated by a comparative study made on the ciliary activity in the frog's mouth under in vivo and in vitro conditions.[1]

Likewise, the present investigation on the turtle and the duck, in which the tracheal cilia are subjected to chemical action through the blood stream under in vivo conditions, justifies the suggestion already made that in approaching problems of ciliary activity in the respiratory tract closer attention should be given to the methods of study in order to maintain as nearly normal physiologic conditions as possible.

The development of the reflection method [2] for the study of ciliary movement in situ, which does not necessitate the addition of foreign

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1. Lucas, A. M.: Comparison of Ciliary Activity Under in Vitro and in Vivo Conditions, Proc. Soc. Exper. Biol. & Med. **30**:501 (Jan.) 1933.

2. Lucas, A. M.: (*a*) Principles Underlying Ciliary Activity in the Respiratory Tract: I. A Method for Direct Observation of Cilia in Situ and Its Application, Arch. Otolaryng. **18**:516 (Oct.) 1933; (*b*) footnote 1.

particles or fluids to the ciliated surface, provided a means by which more accurate information may be obtained regarding the normal physiologic base-line of ciliary activity. The results indicate that the normal condition of palatal cilia in the frog, for example, is one of complete inactivity; that activity can be initiated by localized stimuli such as the addition of fluids, even Ringer's solution, by mechanical means, by drafts of air, by the addition of finely divided particles such as powdered carbon and carmine and by disturbances in the tension of the mucous layer, and that following the cessation of each stimulus the cilia again return to a condition of rest.

The work of McDonald, Leisure and Lenneman [3] revealed for the first time that nervous impulses play a part in the ciliary activity of the frog's palate. Their major conclusion has been confirmed by later investigations, although part of the evidence on which it is based is controversial. Ciliary responses following injections of pilocarpine hydrochloride and epinephrine hydrochloride were carefully reinvestigated by Pohle,[4] whose results were contrary to those of McDonald and his co-workers. Seo's [5] excellent paper published in the same year demonstrated a closely integrated reflex between the dorsal surface of the tongue and the palatal cilia, and the construction of his nerve-cilia preparation contributed definite proof that nervous impulses initiate certain ciliary responses. More recently it has been shown by one of us (Lucas [6]) that the stimulation of nerves produces only an activation of cilia and not their return to the quiescent state, that the sympathetic nervous system does not carry any of the activating fibers as was reported by McDonald, Leisure and Lenneman,[3] and that the palatine nerve receives its cilio-activator fibers by way of the seventh cranial nerve. The threshold value for the cilio-activator fibers in this nerve is fifteen times greater than that for nerve fibers to skeletal muscle, which result, interpreted, on the basis of studies by Bishop, Heinbecker

3. McDonald, J. F.; Leisure, C. E., and Lenneman, E. E.: Neural and Chemical Control of Ciliated Epithelium, Proc. Soc. Exper. Biol. & Med. **24**:968 (May) 1927; New Principles in the Control of the Activity of Ciliated Epithelium, Tr. Am. Acad. Ophth. **33**:318, 1928.

4. Pohle, K.: Zur Frage der nervösen Regulation des Flimmerepithels bei Wirbeltieren, Arch. f. exper. Path. u. Pharmakol. **159**:452, 1931.

5. Seo, A.: Studies on the Nervous Regulation of the Ciliary Movement, Jap. J. M. Sc. Tr., III, Biophysics **2**:47 (March) 1931.

6. Lucas, A. M.: Comparison of Threshold Stimuli in Nerve Cilia and Nerve Muscle Preparations, Anat. Rec. (supp.) **55**:27 (March) 1933; Neurogenous Activation of Ciliated Epithelium, Am. J. Physiol., to be published.

and O'Leary,[7] indicates that the cilio-activator fibers are part of the autonomic nervous system. They have their cells of origin in the brain.

In view of the fact that there now exists considerable information pertaining to the regulation of ciliary activity in the frog's pharynx, certain questions become paramount, such as whether the cilia in the trachea of higher vertebrates may sometimes autonomously cease their vibratile movements, whether acceleration or diminution of rate exists and if so whether this regulation may be of neurogenous origin, and if this proves to be the case whether its functional properties can be correlated with the neurophysiologic scheme tentatively set forth by Bishop, Heinbecker and O'Leary.

In amplification of the last point it may be said that investigations on the responses in the respiratory system of turtles and mammals to stimulation of the vagus have indicated that breathing movements and reflex blood pressure effects are initiated through the large myelinated fibers, that pain is in general transmitted by medium-sized myelinated fibers and that the smooth musculature of the bronchi contracts when nonmyelinated fibers are stimulated, while stimulation of the myelinated fibers of from intermediate to small size produces no apparent response in the lung. Since neither the action of glands nor the movements of cilia were included in these studies it is hoped that the present observations may contribute some data which can be fitted into the neuromotor scheme for the lower respiratory system.

Some preliminary experiments were made on the duck, a warm-blooded animal, but for the sake of greater exactitude it was necessary to use the turtle as the principal experimental animal.

A turtle was fastened to an animal board, the neck extended and the ventral surface of the trachea exposed by a median incision. After parting the thin layer of fascia covering the trachea a narrow strip about 3 mm. wide and 1 cm. long was cut from the median ventral side of the tube. The opening was covered with a thin sheet of celluloid wrapped around the trachea, which provided a transparent

7. Bishop, G. H., and Heinbecker, P.: Differentiation of Axon Types in Visceral Nerves by Means of the Potential Record, Am. J. Physiol. **94**:170 (July) 1930; A Functional Analysis of the Cervical Sympathetic Nerve Supply to the Eye, ibid. **100**:519 (May) 1932. Heinbecker, P.: The Potential Analysis of the Turtle and Cat Sympathetic and Vagus Nerve Trunks, ibid. **93**:284 (May) 1930; Concerning the Nature and Function of Certain Afferent Pathways from the Thoracic Viscera, J. Thoracic Surg. **2**:183 (Dec.) 1932. Heinbecker, P.; Bishop, G. H., and O'Leary, J. L.: Pain and Touch Fibers in Peripheral Nerves, Arch. Neurol. & Psychiat. **29**:771 (April) 1933. Heinbecker, P.; O'Leary, J. L., and Bishop, G. H.: Nature and Source of Fibers Contributing to the Saphenous Nerve of the Cat, Am. J. Physiol. **104**:23 (April) 1933. Heinbecker, P., and O'Leary, J. L.: The Mammalian Vagus Nerve: A Functional and Histological Study, ibid. **106**:623 (Dec.) 1933.

window for observation, care being taken not to injure the blood and nerve supply. The window became air-tight as the tissue fluids and soft connective tissues pressed into the crevices. The cilia were made visible by the employment of an easily adjustable source of light and the specially mounted microscope previously illustrated.[2a] The tissue was illuminated for brief periods only; under continuous illumination the temperature was raised but 0.5 C. The ciliary vibrations were seldom greater than 5 per second and were readily counted against a stop-watch with an error of not more than 15 per cent. Usually the error (as measured by two successive counts and counts by two persons) was less than 10 per cent.

TABLE 1.—*Normal Ciliary Activity in the Trachea of the Turtle*

Brain Destroyed				Brain and Circulatory System Intact											
Experiment 1, Neck Extended				Experiment 2, Neck Extended				Experiment 3, Neck Retracted				Experiment 4, Neck Extended; Trachea Unopened			
Time		Room Temperature, C.	Cilia, Vibrations per Second	Time		Room Temperature, C.	Cilia, Vibrations per Second	Time		Room Temperature, C.	Cilia, Vibrations per Second	Time, Min.		Cilia, Vibrations per Second	
Hr.	Min.			Hr.	Min.			Hr.	Min.						
	0	29	2.3		0	29	4.0		0	30	5.0	0		4.2	
	30		2.2		30		4.0		15		5.0	15		4.5	
	50	30	2.3	1	30		3.6		30		5.0	30		4.2	
1	5		3.0	2	0	33	3.3		45		5.3	45		4.3	
3	5		3.4	2	15		3.5	1	0		5.5				
3	25		2.6	2	30		3.0	1	15		4.8				
3	55		3.1	2	45		3.3	1	30		5.1				
4	25	30	3.2	3	0		3.6	1	45		5.5				
5	25		3.2	3	15		3.0	2	0		5.0				
5	55		3.2	3	30	32	3.0	3	0	31	5.0				
22	25		2.5	3	45		3.4				Av. 5.1				
				4	0		3.8	3	15*		4.6				
				4	15		3.6	3	25		4.4				
				4	30	31	4.0	3	35		4.5				
				4	45		3.4	3	55		4.6				
				5	0		3.4	4	15		4.2				
				5	15		3.6	4	30		4.3				
								4	45		4.3				
								5	0		4.3				
								5	40	32	4.5				
								6	20		4.6				
			Av. 2.8				Av. 3.5				Av. 4.4			Av. 4.3	

* The neck was extended at this time and was retained in this position for the remainder of the experiment.

NORMAL CILIARY ACTIVITY

It was first necessary to determine the physiologic base-line for ciliary movement in the turtle's trachea, especially in view of the fact that it proved to be zero in the frog's pharynx. The rate of ciliary movement in experiment 1, made on a turtle with its brain destroyed, averaged 2.8 vibrations per second (table 1). Observations made at close intervals for the first six hours demonstrated that the cilia never came to rest nor did the rate of movement change suddenly; for example, the total variation was not more than 1.2 vibrations per second.

Experiment 2 was performed in the same way except that the brain was left intact and the head was extended by clamps fastened against the jaws which permitted normal circulation to the nerve tissues. The

data in table 1 show slight variations in the ciliary rate during a period of five and one-fourth hours. The slight fluctuations and their irregularity are probably not functionally significant.

It is conceivable that extension of the neck might in itself be a stimulating factor. Experiment 3 was the same as experiment 2 except that the turtle was permitted to retract its neck as far as possible. The anterior end of the plastron was of necessity removed to construct the chamber in the neck. The average rate of ciliary vibrations for the first three hours was 5.1 per second, and at the end of that period the neck was extended. The average rate for the subsequent three hours was slightly less, 4.4 vibrations per second.

Experiment 4 was designed to show whether the removal of tracheal tissue involved in the construction of the window might be the factor which stimulated the cilia to continuous movement. It is possible to pass the light through the unopened wall and produce a high light on the inner surface. By careful focusing the waves may be observed and the rate determined. The values obtained in experiment 4 (table 1) agree so closely with the general average of those for the opened trachea that the latter may safely be assumed to give "normal" results.

It is concluded, therefore, that the cilia of the turtle's trachea beat incessantly, that at different times and in different specimens the rate may fluctuate at least from 2.2 to 5.5 vibrations per second and that the method of obtaining the data is not a disturbing factor. Failure of cilia to stop in vivo agrees with the observations of Lierle and Moore [8] on excised nasal mucous membrane of mammals.

In many of the experiments a close record was kept of the rate of cardiac and respiratory contractions. There was no indication that the observed variations of ciliary activity in the trachea were associated with movements of either organ. The subsequent experiments indicate also that the ciliary vibrations are not regulated by motor nerves.

ELECTRICAL STIMULATION

Faradic and condensor shock stimulation of the vagus failed to reveal resulting variations in ciliary activity. The observations were made at the time of stimulation while watching the beating cilia through the microscope. Likewise when the electrodes were arranged on each side of the trachea at the level of the tracheal window, strong and weak stimuli failed to produce any alteration of the ciliary rate. Although the vagi were not sectioned in the experiments in which the trachea was observed in situ, the ciliary vibrations on pieces of excised trachea showed no variation in rate. The vagus apparently does not exert the

8. Lierle, D. M., and Moore, P. M.: Effects of Drugs on Ciliary Activity of Mucosa of Upper Respiratory Tract, Arch. Otolaryng. **19**:55 (Jan.) 1934.

same regulatory influence on ciliary as on cardiac movements. This conclusion is supported by Lommel's experiments [9] on dogs in which both vagi were cut without effect on the rate of flow of mucus.

EFFECT OF PILOCARPINE, EPINEPHRINE AND ATROPINE

Three drugs, pilocarpine hydrochloride, epinephrine hydrochloride and atropine, were injected either subcutaneously or into the heart.

TABLE 2.—*Ciliary Activity in the Trachea Following the Injection of Sympathomimetic Drugs*

Pilocarpine Hydrochloride				Epinephrine Hydrochloride (1:1,000 Solution)				Atropine (1% Solution)			
Time		Cilia, Vibrations per Second	Injections and Comment	Time		Cilia, Vibrations per Second	Injections and Comment	Time		Cilia, Vibrations per Second	Injections and Comment
Hr.	Min.			Hr.	Min.			Hr.	Min.		
			Subcutaneous injection into hindleg		0	3.7	Average of 4 counts; 1 cc. subcutaneously		0	3.8	Average of 3 counts; 1 cc. subcutaneously
	0	2.9			15				15	3.5	
	15	3.6			30	4.4			30	4.0	
	30	3.9			45	4.2			35		1 cc. subcutaneously
	45	3.4		1	15				45	4.0	
1	0	45.3	Copious discharge from nose and eyes	1	20		0.5 cc. subcutaneously	1	0	3.9	
				1	30	4.0		1	20	3.9	
1	15	4.3		1	45	4.1		2	0	4.0	
1	30	4.0		1	50		0.5 cc. subcutaneously	3	0	4.0	
2	0	4.0		2	0	4.2		3	5		1.5 cc. subcutaneously
2	5		Second subcutaneous injection	2	15	4.3		4	30	3.6	
				2	20		1 cc. subcutaneously	4	35		1.5 cc. into the caval vein
2	15	3.9		2	30	4.0					
2	35	4.0		2	45	4.2		4	45	4.0	
2	40		Third subcutaneous injection	3	0	4.0		5	0	3.9	
				3	15	3.7		5	15	4.0	
3	5	4.5						5	30		1.5 cc. into the heart
3	15	4.2						6	10	3.6	
3	30	4.0						6	35	3.4	
3	45	4.3						7	0	3.6	
		Av. 3.9				Av. 4.1				Av. 3.8	

Pilocarpine produced a copious flow of secretions from the eyes, nose and mouth. Yet this stimulation evidenced by the increased discharge of the glands failed to cause any variation in ciliary activity (table 2). This was surprising in view of the intimate functional relation between the production of a layer of mucus and its propulsion. The average rate of vibration throughout the period of the experiment agreed closely with the rates given in table 1.

Epinephrine injected subcutaneously also failed to influence the rate of ciliary movement. The fluctuations were small and usually no greater than errors in making the counts. Lierle and Moore [8] applied epinephrine hydrochloride in a solution of 1:1,000 to the washed, exposed

9. Lommel, F.: Zur Physiologie und Pathologie des Flimmerepithels der Atmungsorgane, Deutsches Arch. f. klin. Med. **94**:365 (Oct.) 1908.

nasal mucosa of the living guinea-pig and observed in vivo that the cilia were stopped within five minutes. Hill [10] previously reported that a concentration of epinephrine which caused uterine contraction when applied to the excised trachea had no effect on the cilia.

Atropine showed the same inability to modify the frequency of ciliary vibrations. The average rate was less than with epinephrine.

TABLE 3.—*Ciliary Activity in the Trachea of the Turtle Following the Injection of Morphine and Alcohol*

Morphine and Hydrochloric Acid (1:1,000)					Ethyl Alcohol, 10%				
Time		Cilia, Vibrations per Second	Heart Beats per Minute	Injections and Comment	Time		Cilia, Vibrations per Second	Heart Beats per Minute	Injections and Comment
Hr.	Min.				Hr.	Min.			
	0	4.5	25			0		34	
	45	4.2	47			15		35	
1	0	4.5	73	1.5 cc. subcutaneously		45	4.4	40	
					1	5		52	
1	15	4.2	68		1	15			10 cc. into stomach
1	30	4.4	28						
1	45	4.3	48		1	45	4.2	50	20 cc. into stomach
2	0		24						
2	15	4.2	50		2	10	4.0	55	
2	30	4.0	40		2	25	4.1		15 cc. into stomach (mucus viscous)
3	30	4.1	85	1.5 cc. subcutaneously					
3	45	3.9	90		2	45	4.4		
4	0	4.0	84		3	0			15 cc. into stomach
4	15	4.0	88						
4	30	3.9	88	1.5 cc. subcutaneously	3	10	4.3	58	
					3	30	4.5	59	
4	45	3.8	90		3	40			10 cc. into stomach
5	0	3.8	84						
5	20	4.0	86	1.5 cc. subcutaneously	3	45	4.1		
					4	0	4.5	50	10 cc. into stomach
5	40	4.2	88						
5	50	4.0	89	1.5 cc. subcutaneously	4	15	4.4	50	
					4	45	4.2	48	
6	20	4.0	84		5	5	4.5	46	
6	30			2 cc. into the heart	5	20	4.5	40	Total: 80 cc.
6	50	4.0	88				Av. 4.3		
7	0			3 cc. subcutaneously					
7	15	4.0	92						
7	30	4.2	86						
7	45	3.9	87						
		Av. 4.1		Total: 12.5 mg.					

Atropine perhaps thickens the mucus and decreases its flow, while pilocarpine has the opposite effect. Yet under the action of both drugs the ciliary rate remains approximately the same. The physical alteration of the secretion undoubtedly affects the efficiency and speed with which the layer mucus moves along, but the motility of the agents responsible for this clearance has not been altered. Lierle and Moore [8] found that in twelve of thirteen trials a 1 per cent solution of atropine applied to excised nasal mucosa for five minutes did not affect the rate of ciliary movement.

10. Hill, L.: The Ciliary Movement of the Trachea Studied in Vitro, Lancet **2**:802 (Oct. 20) 1928.

EFFECT OF MORPHINE AND ALCOHOL

Hach [11] timed the passage of particles along the dog's trachea in vivo. The ciliary rate determined on the basis of the rate of passage of pollen of colored lycopodium from the time of injection into one of the small bronchi until its appearance at a tracheal opening previously prepared averaged 36 microns per second in the animal anesthetized with morphine. When Hach repeated the same experiment the next day after the effects of the morphine had disappeared the rate of flow of mucus was 76 microns per second, and after the injection of small doses of caffeine (from 0.006 to 0.008 Gm. per kilogram of body weight) the rate averaged 227 microns per second. Worley [12] found that of several drugs applied to excised gills of Anodonta and to ciliated mucosa of the frog only caffeine produced a strong initial acceleration of ciliary movement. Lommel's experiments [9] with morphine, also under in vivo conditions, showed no effect on the tracheal flow of mucus. His method differed from Hach's in that after the drug was administered the dog was killed, the trachea excised and the activity studied by application of particles to the ciliated surface. This procedure was not as truly an in vivo method as was Hach's. In Umeda's experiments [13] on the excised trachea of the ox no variation in the ciliary rate was found following the administration of morphine hydrochloride. This was contrary to earlier investigations by Ishikawa [14] who used tissue culture methods with ciliated epithelium of the frog.

A compilation of data from one of two experiments is given in table 3. The heart was exposed through a small opening made in the plastron. Increased heart action occurred in both experiments and probably indicates that the quantity of drug used was sufficient to obtain at least initial effects of morphine. It was not enough to produce deep anesthesia. The cilia showed no significant variation, and the average rate agreed closely with the normal value. In one experiment the slowing of the passage of mucus may have been due to slightly increased viscosity. Toward the end of the experiment a thick layer of mucus accumulated so that the cilia could no longer be seen, but the last record that could be taken showed that the rate of the ciliary vibrations had not decreased. The accuracy of Hach's data cannot be questioned.

11. Hach, I. W.: Zur Frage über die Flimmerbewegung in Organismus homoiothermer Tiere, Ztschr. f. d. ges. exper. Med. **46**:558, 1925.

12. Worley, L. G.: The Dual Nature of Metachronism in Ciliated Epithelium, J. Exper. Zoöl. **69**:105 (Oct.) 1934.

13. Umeda, T.: A Study on Ciliary Movement of Ox Trachea, Acta dermat. **14**:645, 1929.

14. Ishikawa, S.: Kultur der Epithelien in Vitro: II. Studien über die Flimmerepithelien in Vitro, Acta dermat. **9**:363, 1927.

His results are averages for many animals. Perhaps they can be interpreted, however, in terms of changes in flow and viscosity of mucus, which are subject to variations independent of ciliary vibrations. Caution must be exercised, moreover, in applying information gained from observations on the turtle to the mammal, especially in view of the fact that the mechanism of ciliary regulation in two animals as closely related as the frog and the turtle exhibits very different physiologic properties.

Lommel [9] reported from experiments on dogs forced to drink quantities of dilute alcohol that vibration of the cilia (as judged by the rate of flow of mucus) slowed greatly and usually stopped completely. The effects of alcohol on excised horse trachea were investigated by Hill,[10] who found that "a weak percentage of alcohol has no effect and a strong one retards ciliary movement." Umeda [13] noted that dilute ethyl alcohol applied to explants of ciliated epithelium of the frog was temporarily able to accelerate the ciliary movement slightly. Carter [15] found that the beat of the velar cilia of the nudibranch veliger soon weakened when the larva was immersed in 6 per cent alcohol. The effect of alcohol is important clinically in this regard: Alcoholism has been cited in discussions of lobar pneumonia as being the most potent predisposing factor.[16]

As a check on the in situ observations reported here, a few experiments on the excised trachea of the turtle were made. Dilutions of alcohol of 10 per cent and less permitted ciliary movement for at least a minute when the layer of mucus was not previously removed with saline solution, and 20 per cent alcohol under the same conditions stopped the cilia between thirty seconds and one minute. Therefore, the cilia of the excised trachea of the turtle act toward alcohol in the same general way as the excised trachea of a mammal as reported by Hill.

The injection of alcohol into the stomach by means of a tube and its subsequent absorption did not throughout the course of five hours and twenty minutes affect the rate of ciliary movement in the trachea. The experiment was repeated on a second turtle, and the results were identical. One received a total of 80 cc. of 10 per cent alcohol and the second 60 cc. Almost with the first injection the animal lost muscular tone and the skin blanched. Signs of recovery were followed by another injection; so the animal was well narcotized during a five hour period. There seemed to be a tendency toward a drying effect of the mucus and of the tracheal tissues; this occurred in spite of the fact that the light was on only for short periods and that during the intervals the exposed

15. Carter, G. S.: On the Nervous Control of the Velar Cilia of the Nudibranch Veliger, Brit. J. Exper. Biol. **4**:1 (Sept.) 1926.

16. Osler, W.: Principles and Practice of Medicine, ed. 11, New York, D. Appleton & Company, 1930, p. 82.

tissues were covered with a pad moistened in saline solution. It may be that alcohol is responsible for a dehydrating effect; if so, this may account for Lommel's results. It is certain, however, that the doses of alcohol given in these experiments do not result in any dysfunction of ciliary activity.

Since the results of in vivo studies of ciliary movement on the turtle led to many conclusions contrary to those derived from in vitro experiments and since the ciliary beat in vivo was unaffected by the reagents, drugs and stimuli applied, it seemed advisable to subject the animal to changes of temperature to make sure that it did not differ in this reaction. Previous studies on this subject as applied to the excised trachea have already been reviewed.[17] Lommel [9] noted that in dogs required to breathe subzero air there was no slowing of ciliary movement.

The experiments already described and presented in table 1 were made on turtles kept at a room temperature of from 29 to 32 C. The average ciliary rate was about 4 vibrations per second. When the animals were cooled no record of ciliary movement was obtained below 6 C. At 6 C. the rate was 0.9 vibration per second; at 10 C., 1.4 vibrations per second, and at 20 C., 3 vibrations per second. The cilia of the turtle's trachea react, therefore, to changes of temperature in accord with the results of previous investigators.

In experiments on ducks the neck was prepared in the same way as in the case of the turtles, except that the ducks were anesthetized with sodium amytal. Six ducks were used. The chief difficulty involved in the use of a warm-blooded animal is the condensation of moisture on the under wall of the transparent covering for the tracheal window, if the chamber is not kept heated. No records of rate were obtained for the duck. It was clearly evident that the duck and the turtle are similar to the extent that the cilia of the trachea of both vibrate continuously.

COMMENT

We have suggested previously [18] that the normal mucous sheet is composed of two layers: an inner one of fluid surrounding the cilia and an outer one of mucus which carries débris and foreign particles. The inner layer flows with the vibrating of the cilia, but the outer layer may or may not respond to the beating of cilia against its under surface. The application of this information to the results obtained by Lommel [9] and Hach [11] indicates that the rate of flow of mucus cannot be used as a reliable measure of ciliary movement.

17. Lucas, A. M.: Ciliated Epithelium, in Cowdry, E. V.: Special Cytology, New York, Paul B. Hoeber, Inc., 1932, p. 408.

18. Lucas, A. M., and Douglas, L. C.: Principles Underlying Ciliary Activity in the Respiratory Tract: II. Mucous Clearance in Man, Monkey and Other Mammals, Arch. Otolaryng. **20**:518 (Oct.) 1934.

The present results indicate that the injection of various narcotics and drugs, most of which usually elicit a response in conjunction with the nervous system, does not modify the ciliary movement in the trachea. This stands in contrast to numerous experiments on substances applied to excised tissues in which the vibrations of the cilia are slowed or stopped.

Particularly significant is the work of Lierle and Moore [8] in which the reflection method was employed to the exposed nasal cilia of the guinea-pig under in vivo conditions. Instead of applying the drug to the ciliated epithelium through the blood stream they placed it directly on the mucosa following a previous washing with Ringer's solution; under such conditions the cilia were affected in general in about the same way as when excised tissue is subjected to similar treatment.

In view of the fact that the present experiments indicate that the epithelium is not innervated by motor nerves, the differences between the effects of drugs applied to the surface and those of drugs carried by the blood stream may be due merely to a difference in the concentration of the reagents employed in the two ways. The concentration would obviously be much less when the drug is carried by the blood than when it is applied directly. There is also the possibility, of course, that an equal concentration of the drug causes a greater reaction when applied to the cilia directly than when the point of entry is through the base of the cell.

The theory of the absence of motor innervation of the tracheal epithelium is supported by the neurologic studies of Larsell [19] and Larsell and Dow [20] on man. They found nerve fibers having characteristic motor endings ramifying among the submucous gland cells. On the other hand, only nerves having endings of a sensory type were found terminating in the epithelium. Thus the conclusions reached in the neurologic studies and the results of the present physiologic experiments are in agreement. The contribution of Florey, Carleton and Wells [21] on the physiology of secretion of mucus in the trachea of mammals confirmed the neurologic studies. These authors observed that pilocarpine and stimulation of the peripheral or central end of a vagus activated the subepithelial glands but did not affect intra-epithelial glands—the goblet cells—whereas an irritant (oil of mustard) applied directly caused a marked emptying of the goblet cells. Although they observed the cilia

19. Larsell, O. P.: Nerve Terminations in the Lung of the Rabbit, J. Comp. Neurol. **33**:105 (June) 1921; The Ganglia, Plexuses and Nerve Terminations of the Mammalian Lung and Pleura Pulmonalis, ibid **35**:97 (Dec.) 1922.

20. Larsell, O., and Dow, R. S.: The Innervation of the Human Lung, Am. J. Anat. **52**:125 (Jan.) 1933.

21. Florey, H.; Carleton, H. M., and Wells, A. Q.: Mucus Secretion in the Trachea, Brit. J. Exper. Path. **13**:269 (June) 1932.

by reflected light they did not record whether the acceleration of the passage of mucus due to the injection of pilocarpine was due to an increased flow of mucus alone or whether the vibrations of the cilia were also accelerated. The results of the present investigation indicate that the cilia take no part in the accelerated passage and that the acceleration must be explained, therefore, as due to lessened viscosity of the mucus which renders the ciliary beating more effective.

If one applies these conclusions to the problems of the neurophysiologist, it seems probable that in the separation of nerve-potential components producing motor responses in the respiratory system one may anticipate that within some particular potential group the activating nerve fibers for the glands will be identified, while, on the other hand, it seems that the expectation is small of finding cilio-activator nerve fibers.

SUMMARY

The normal rate of ciliary vibrations in the trachea of the turtle studied in situ at temperatures of about 30 C. was from 2.2 to 5.5 per second.

No change in rate observable under in vivo conditions occurred following the injection of pilocarpine, epinephrine, atropine, morphine or alcohol, nor did electrical stimulation of the vagus or the tracheal tissue produce any effect. This condition is in contrast to results often obtained when chemical substances are applied directly to the ciliated surface.

Association of these facts with the results of physiologic studies of the vagus nerve, of secretion of mucus in the trachea and of innervation of glands by previous investigators suggests that the motor nerves to the trachea affect only the rate at which the mucus is poured forth and do not change the rate at which the cilia vibrate in propelling it along.

Wide variations, which have been described, in the rate of flow of mucus are probably due, therefore, to physical and perhaps associated chemical changes in the layer of mucus which correspondingly vary the efficiency of its propulsion by the cilia and do not represent a modified rate of ciliary movement.

PATHOLOGY OF OTOSCLEROSIS

WITH REPORT OF CASES

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In considering the pathology of otosclerosis one finds in the literature a large number of reports [1] and considerable differences of opinion as to the interpretation of the observations. Most of the discussions have centered about the changes in the bony capsule of the labyrinth. However, Gray [2] has written several times about changes in the eighth nerve. He has expressed the belief that he has found a localized loss in the neurokeratin of the medullary sheaths and a similar absence in the neurilemma of the cochlear nerve. These changes were absent in the vestibular division. Other observers have found degeneration in the organ of Corti, the spiral ganglion and the eighth nerve.

In considering these changes one must remember the normal difference between the two divisions of the eighth nerve. The fibers of the vestibular branch are normally larger, more distinct and more easily seen. The neurokeratin is visible, whereas in the cochlear division it is not distinguishable. The neurilemma is normally seen in both branches. In the cochlear portion the fibers of the endoneurium are more abundant. A study of the five otosclerotic bones considered in this present report did not reveal any of the pathologic changes in the eighth nerve described by Gray and others. The neurilemma was as definite in the cochlear division as in the vestibular. Thus it may still be questioned whether changes characteristic of otosclerosis occur in the eighth nerve. Degeneration in the organ of Corti and the spiral ganglion has been found in cases of otosclerosis, but the degeneration would not necessarily have to be on the same basis as the otosclerotic bony foci. It would seem that to prove reasonably the connection between the pathologic processes in the several parts of the ear there should be a considerable number of cases showing similar changes; the corresponding parts in control cases should not show these alterations.

Returning to the consideration of the changes in the bone, one finds otosclerosis recorded in a child as young as 1 year old, and recently

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1. American Otological Society: Otosclerosis, vols. I and II, 1929.

2. Gray, A. A.: The Otosclerosis Problem: Reports of Two Cases Examined Pathologically, J. Laryng. & Otol. **49**:629 (Oct.) 1934.

Guggenheim [3] found what he believed to be an early stage of the disease in a 7 months fetus. Some of the theories as to the development of otosclerotic bone are: (1) vascular constriction (O. Mayer); (2) altered vascularization due to an inflammatory process in the tympanic cavity (Siebenmann); (3) chronic local infection in the mucoperiosteum of the tympanum, such as that following otitis media (Fraser); (4) chronic inflamation (Manasse); (5) venous stasis (Wittmaack); (6) a gradually increasing defect in the vasomotor mechanism which governs the nutrition of the ear (Gray); (7) development from the part of the labyrinth last ossified (Pierce), and (8) regression (Guggenheim).

Practically all observers agree that there is probably a hereditary factor in these cases and that the condition is usually bilateral. The various general factors which may influence the condition are syphilis, tuberculosis, gastro-intestinal toxemia, toxemia from any focus of infection, alterations in the function of the glands of internal secretion, faulty diet (lack of vitamins), arteriosclerosis and pregnancy.

The change in the bony labyrinth in otosclerosis is characterized by its occurrence in localized patches, termed foci. No other bone disease is exactly like it, although several are similar to it except for the focus formation. The commonest site for a focus is just anterior to the footplate of the stapes. This has been called the "site of predilection," and it is here that the fissula ante fenestram is found. Other foci are found, however, (*a*) surrounding one side of the cochlea and often extending to the internal auditory meatus; (*b*) about the round window, and (*c*) about the semicircular canals. The focus of otosclerosis is usually found to consist of a spongy type of bone with vascular, marrow-like cavities. The majority of cells about the vessels in these spaces are probably osteogenic. The bone cells are irregularly laid down, haversian systems being absent or infrequent. Osteoblasts and osteoclasts are frequently seen in the focus; in some cases they are scarce, in other cases more plentiful. While some observers have found these cells at the border of the focus adjacent to the normal bone, others have not seen them in this location. At the site of predilection the focus frequently develops toward the stapedial foot plate and may invade and replace more or less of it. It may even form an exostosis toward the cavum tympani. Vessels from the focus often connect with the overlying mucous membrane of the cavum tympani. The subepithelial tissue may be quite vascular or it may be thickened and fibrous. In many foci finger-like projections, usually surrounding a vessel, extend out from the focus into the surrounding bone. The foci appear to develop at the junction of the enchondral layer of the bony labyrinth and the periosteal layer. As the focus develops the periosteal, enchondral and endosteal layers

3. Guggenheim, L. K.: Otosclerosis in a Seven Months Fetus, Ann. Otol., Rhin. & Laryng. **42**:690 (Sept.) 1933.

become involved. The focus takes a striking bluish-red stain with hematoxylin and eosin and can often be recognized without the microscope. The remnants of cartilage often seen normally in the bony labyrinth are not found in the focus.

Two main views have been held as to the enlargement of the foci. The first view postulates that the normal bone is absorbed by osteoclasts and is replaced by the otosclerotic bone. The second view is that the focus is a tumor-like formation and that it destroys the normal bone as it develops. The frequent absence of osteoclasts at the edge of the focus speaks against the first view, and the absence of a common tumor cell argues against the second view. It is possible that the bone adjacent to the focus is prepared by chemical action and that the focus enlarges by sending out the finger-like processes into the chemically altered bone and gradually replacing it. Some investigators have held that the otosclerotic process is inflammatory, while others, failing to find round cells or lymphoid cells, have denied this. Otosclerosis may be found alone, or it may be associated with other conditions, such as acute or chronic otitis media, congenital malformation, cretinism, osteitis fibrosa, neurofibroma of the eighth nerve, fragile bones and blue sclera, tuberculosis, leukemia and Gaucher's disease.

I wish to present briefly observations on five temporal bones showing otosclerosis from four patients. Four of the bones have previously been reported [4] on in detail. Of the four patients, two were men and two women. The youngest patient was a woman who died at the age of 20; the oldest was a man who was 70 years old at the time of death. The latter patient was the only one whose condition was diagnosed as otosclerosis during life.

REPORT OF CASES

CASE 1 (Bone 1).—Ta, a woman aged 37, died of intracranial complications following acute suppurative otitis media. As far as could be ascertained there had been no previous history of deafness or aural trouble.

Only the right, the involved, ear was furnished for microscopic examination. Study of the bone revealed infection of the pneumatic spaces and, in addition, a definite focus at the site of predilection just anterior to the footplate of the stapes. The footplate was not involved, but the focus replaced the vestibular lip of the joint. The fissula ante fenestram was absent except for its notchlike beginning at the vestibule. From the anterior joint of the stapes the connective tissue was seen to extend around the tympanic border of the focus in a fibrous-like layer. This was nonvascular and apparently noninflammatory. The focus bone was vascular but had few osteoblasts and practically no osteoclasts. In this case both otosclerosis and acute suppurative otitis media were present.

CASE 2 (Bones 2 and 3).—Fe, a woman aged 20, died of otitic intracranial complications. Her history was that at the age of 5 she had scarlet fever which had left her with a chronically discharging left ear associated with moderate

4. Hagens, E. W.: Otosclerosis, Arch. Otolaryng. **12**:273 (Sept.) 1930.

deafness. No deafness was noted in the right ear. An acute exacerbation of the condition in the ear led to fatal complications.

Examination of the left ear revealed considerable infection but, in addition, several foci of otosclerosis. The foci were found at the site of predilection, about the cochlea extending from the apex around to the internal auditory meatus and about the posterior semicircular canal. The first-mentioned focus had invaded the stapes and replaced nearly all of the footplate. In addition, the bone was much enlarged, forming hyperostosis. A distorted anterior joint ligament could be seen, and connective tissue from this extended around the tympanic border of the focus as a fibrous, nonvascular, noninflammatory layer. The fissula ante fenestram was not seen. Osteoblasts with new bone underlying and osteoclasts were seen in the focus. The focus was quite vascular. This bone revealed three foci of otosclerosis. Thus chronic suppurative otitis media and otosclerosis

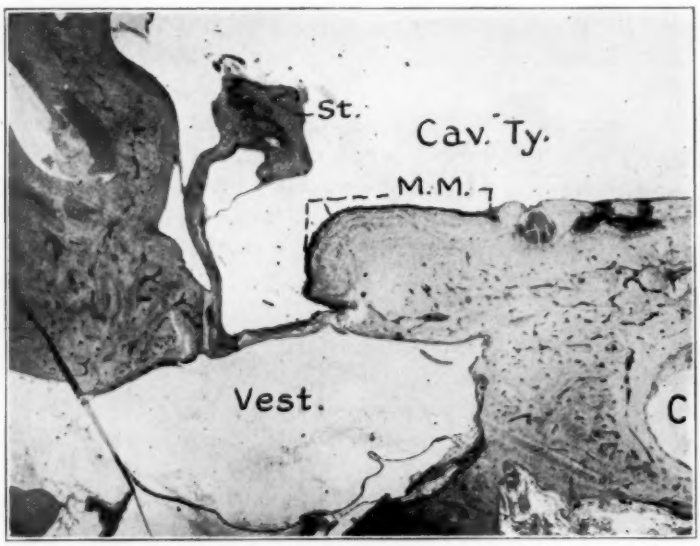

Fig. 1.—A section from the right ear, showing the normal niucous membrane (*MM*) overlying the bone in the region of the stapes (*St.*). *Cav. Ty.* indicates the cavum tympani; *Vest.*, the vestibule; and *C.*, the cochlea. Magnification, × 14.

occurred together. Clinically, the deafness in this ear would be sufficiently explained by the chronic suppurative otitis media, the otosclerosis being entirely masked by the otitis.

The right ear revealed a focus at the site of predilection, one extending around the cochlea on the left side, one about the round window and a fourth about the medial crus of the horizontal semicircular canal. The focus in the region of the stapes did not actually invade this ossicle but did form definite hyperostosis about it. The appearance of the focus was similar to that on the left side. The mucous membrane overlying the focus on its tympanic side was thickened by dense, fibrous tissue. The fissula ante fenestram was not seen. In this bone there were four foci, and yet the history did not reveal aural infection or deafness on this side.

Case 3 (Bone 4).—W, a man aged 70, died of organic heart disease. Dr. Shambaugh diagnosed otosclerosis seven years before the patient's death.

There was a history of severe bilateral deafness and tinnitus for a number of years. Both temporal bones were obtained, but the right one was so altered during the preparation that only the left one could be studied accurately.

Examination of the left temporal bone revealed a single focus at the site of predilection. The focus involved a considerable part of the stapedial footplate and anterior crus, producing hyperostosis. An irregular band of connective tissue simulated the anterior joint and continued over the tympanic surface of the focus. In places blood vessels were seen in the connective tissue. The bone of the focus was quite sclerotic, the marrow-like vascular spaces being small. Osteoblasts and osteoclasts were not seen. The fissula ante fenestram could not be recognized. This case represents the older, more sclerotic type of focus.

CASE 4 (Bone 5).—Mi, a man aged 35, died of syphilitic meningitis. A gumma was found in the right cerebral hemisphere, and the brain showed marked edema.

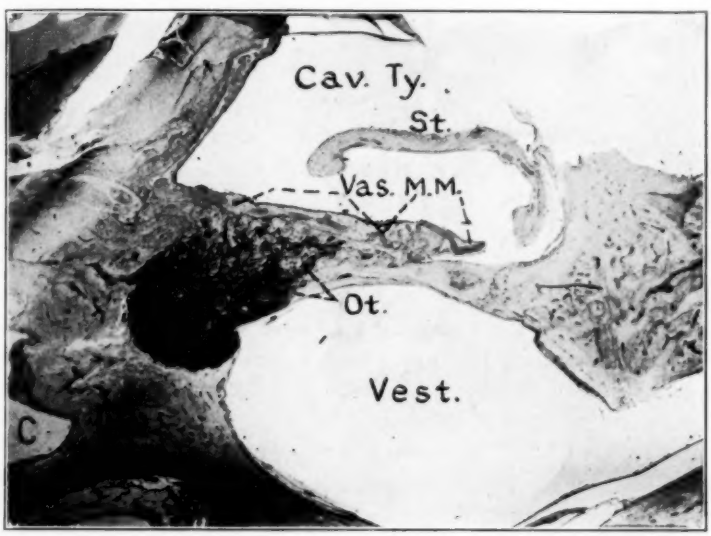

Fig. 2.—A section from the left ear, showing vascular mucous membrane (*Vas. M.M.*) overlying the focus of otosclerosis (*Ot.*) in the region of the oval window and stapes (*St.*). *Vest.* indicates the vestibule; *C*, the cochlea, and *Cav. Ty.*, the cavum tympani. Magnification, × 14.

There was no history of deafness or aural trouble except frequent otalgia with tinnitus as a child. There was no history of deafness in the family. Severe headache with pain back of the eyes had been present since childhood.

Examination of the temporal bone revealed absence of suppuration on each side. The right temporal bone revealed no otosclerosis, and the tympanic mucous membrane overlying the stapes and surrounding bone was normal (fig. 1). The fissula ante fenestram was easily identified. The left temporal bone revealed a focus at the site of predilection. The stapes and its foot-plate were not involved. The focus was quite vascular; in one place a large finger-like vessel extended into the adjacent bone in the direction of the processus cochleariformis. Only a few osteoblasts and osteoclasts were seen. The fissula ante finestram was indicated only by a small notch at the vestibule. Of considerable interest was the tympanic

mucous membrane overlying the focus and present to a lesser extent about the crura. Examination revealed a greatly thickened subepithelial layer which was extremely vascular (fig. 2). Larger vessels with thin walls almost filled this layer. No definite evidence of past or present inflammation could be seen. This, then, represented the pathologic basis for the clinical Schwartze sign. Unfortunately, no record of the appearance of the drum membranes was made on the chart.

The interest in this case lies in the finding of unilateral otosclerosis with a definite opportunity for comparison of the two sides. There was no history of suppurative otitis media. If otalgia meant a lesser type of involvement and if both ears were involved, as might be expected, nevertheless otosclerosis developed only on one side, and on this side the tympanic mucosa was extremely vascular while on the other it was normal.

SUMMARY

A summary of the findings in this series of bones reveals that most of the points made in the literature are demonstrated. All the foci stained prominently with hematoxylin and eosin. In a previous article [5] I attempted to show that this may be due either to the unusual attraction of the focus bone for the mordant in the stain or to the presence of iron. A number of observers have thought the peculiar staining was due to the excess of calcium in the focus, but this could not be proved. With regard to the fissula ante fenestram, the foci were all too far advanced for one to tell whether they had started from this structure or had engulfed it in their development. The close connection of the overlying thickened tissue on the tympanic side of the focus would lead one to believed that the thickened membrane, vascular or not as the case may be, was related to the focus and was not associated with otitis media. This belief is further strengthened by the observations in case 4 and by the presence of foci far from the cavum tympani as in case 2. Also in case 2 there was more otosclerotic involvement in the nonsuppurative ear than in the suppurative ear.

Finally, one may say that no theory of etiology seems adequate at present. No descriptive term for the pathologic process is wholly satisfactory, and the name otosclerosis, while unsatisfactory, is still the best known.

30 North Michigan Avenue.

5. Hagens, E. W.: Otosclerosis: Staining and Chemical Reactions, Arch. Otolaryng. **13**:824 (June) 1931.

FISSULA ANTE FENESTRAM

ITS FORM AND CONTENTS IN EARLY LIFE

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The fissula ante fenestram has come to be regarded as a constant feature of the human ear and is believed to occur typically as a channel within the bone containing an avascular connective tissue. In early corrosion preparations described by Hyrtl[1] in 1845 and by Siebenmann[2] in 1890 only the medial end of the fissula was demonstrated, and in their figures it appears as a macroscopic projection of the anterior portion of the vestibule. Later, Mayer[3] (1917) and others have described and figured the full structure with its termination on the wall of the tympanic cavity. They have emphasized that it is a fissure-like band of connective tissue within bone, and, because of its location between the cochlear and the canalicular portion of the capsule, they have maintained that it serves as a synchondrosis between them, absorbing the strains incident to growth.

From a study of forty series of sections, we find that important departures from this simple plan, both as to form and as to structure, are frequent; yet both the common and the irregular forms can be arranged in a graded series, the differences between the near members of which are explicable on the basis of successive histologic change. To discuss these various kinds of fissulae, some of which are no longer channels through the otic capsule, is the purpose of this paper.

MATERIAL AND METHODS

The material on which this investigation is based is part of the collection in the department of otolaryngology at Northwestern University Medical School and was

From the Department of Anatomy, Northwestern University Medical School.

Contribution 210 from the Anatomical Laboratory of Northwestern University Medical School; reported at the Philadelphia meeting of the American Association of Anatomists; abstracted in Anat. Rec. (supp.) **58**:2 (March 25) 1934; reported by Dr. J. Gordon Wilson at the meeting of the Collegium Oto-Rhino-Laryngologicum Amicitiae Sacrum in Stockholm in September 1934.

1. Hyrtl, Joseph: Vergleichend-anatomische Untersuchungen über das innere Gehörorgan des Menschen und der Säugethiere, Prague, F. Ehrlich, 1845.

2. Siebenmann, Friedrich: Die Korrosions-Anatomie des knöchernen Labyrinthes des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1890.

3. Mayer, Otto: Untersuchungen über die Otosklerose, Vienna, Alfred Hölder, 1917.

placed at our disposal through the courtesy of Dr. J. Gordon Wilson.[4] All the temporal bones were fixed in a dilute solution of formaldehyde, U. S. P., and decalcfied in a solution of nitric acid in a dilute solution of formaldehyde; they were sectioned (at a thickness of from 20 to 30 microns) by the pyroxylin method and stained chiefly with hematoxylin and eosin. The models were prepared by the Born method [5] and represent the nonosseous tissues enclosed by the fissula, together with enough of the space of the tympanic cavity and of the vestibule to reveal the relations at both orifices (compare figures 1 and 2). Where connective tissue originally contained within the fissula has been replaced by cartilage, marrow or sclerotic bone, such contents are shown in the model as representing the probable extent of the original fissure within the bone.

The fissula, according to Bast [6] (1933), is well established in the 14 week fetus (100 mm., crown-rump length), although its future site can be discerned at a much earlier stage (50 mm.). The vestibular end of the fissula arises through an invasion of mesenchymal tissue from the vestibular wall into the precartilaginous otic capsule at the angle of junction of the vestibule and the scala vestibuli. At the tympanic end the mesenchymal tissue forms a vascular bud which, in invading the precartilage, meets the evagination from the vestibule and thus forms a channel through the lateral capsular wall. In both instances, along the course of the advancing mesenchyma, precartilage undergoes dedifferentiation. When the precartilage of the capsule changes to cartilage,

4. This report represents one phase of an investigation conducted under the auspices of the Committee on Research of the American Otological Society of which Dr. Wilson is a member. Dr. Wilson has shown great interest, in a supervisory capacity, in all of the aspects of this study. The models on which the present study is based were prepared by Dr. John Martin Jr. in partial fulfilment of the requirements for the degree of Master of Science.

5. The tracings of the Born wax-plate models were prepared with an Edinger projection apparatus at a magnification of 125 diameters (except figure 1, which was magnified 50 diameters). Selected tracings from each series, representing the illustrative levels in the models, were used for the histologic figures. The drawings of the models and of the sections were reduced one-half in preparation (the former by direct measurement, the latter by the use of the precision pantagraph); they were further reduced (to approximately 21 diameters) in reproduction. The auxiliary drawings (*2a* and *2b*, *6a* and *6b*, etc.) of parts of the models were made from measurements taken on the models and, except in two instances (*2a* and *2b*), represent a plane different from that of the sections. They serve to show the form of the fissula at the extremities or at the midpoint. The photomicrographs were made at a magnification of 56 diameters and reduced one-fourth in reproduction. The specimens from the fetuses are from the temporal bone of the right side, and all others from the left. Miss Marjories Hoag prepared all of the drawings of the models and assisted in the preparation of the histologic figures while following a course of study in this laboratory.

6. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1 (July) 1933.

the fissure remains as a streak of areolar tissue, which is subsequently converted into the adult type of connective tissue. The cartilage immediately surrounding the connective tissue persists after the capsule as a whole is osseous and may be found, often at the two extremities, even in adult ears. More commonly, we believe, it is evident only at the vestibular extremity, incompletely following the course of the fissular connective tissue. It is invariably continuous with the cartilage lining the vestibular window; likewise, it is persistent throughout life from the embryonic stage onward. As the tympanic portion extends medially and the vestibular part anteriorly, they join at an acute angle and are related to each other in such a way that the form of the whole structure is that of the letter C.

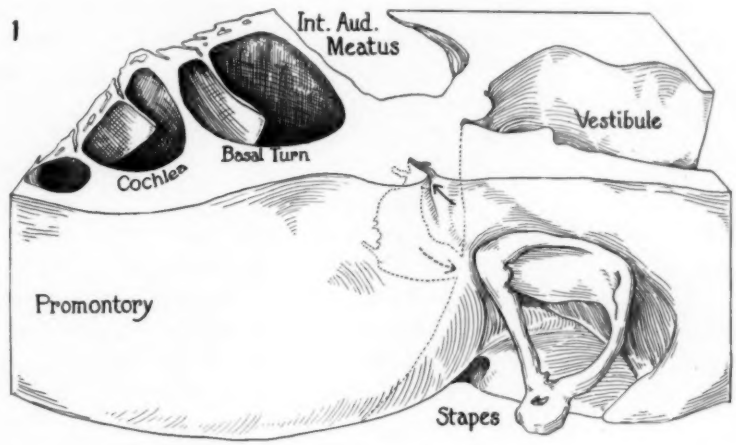


Fig. 1.—Craniolateral aspect of a model of part of the osseous labyrinth, showing the position and relations of the fissula ante fenestram (adult, aged 18; see also figures *2* and *3*). The upper arrow points into the tympanic orifice of the fissula; the lower arrow points out of the vestibular orifice; × 12.

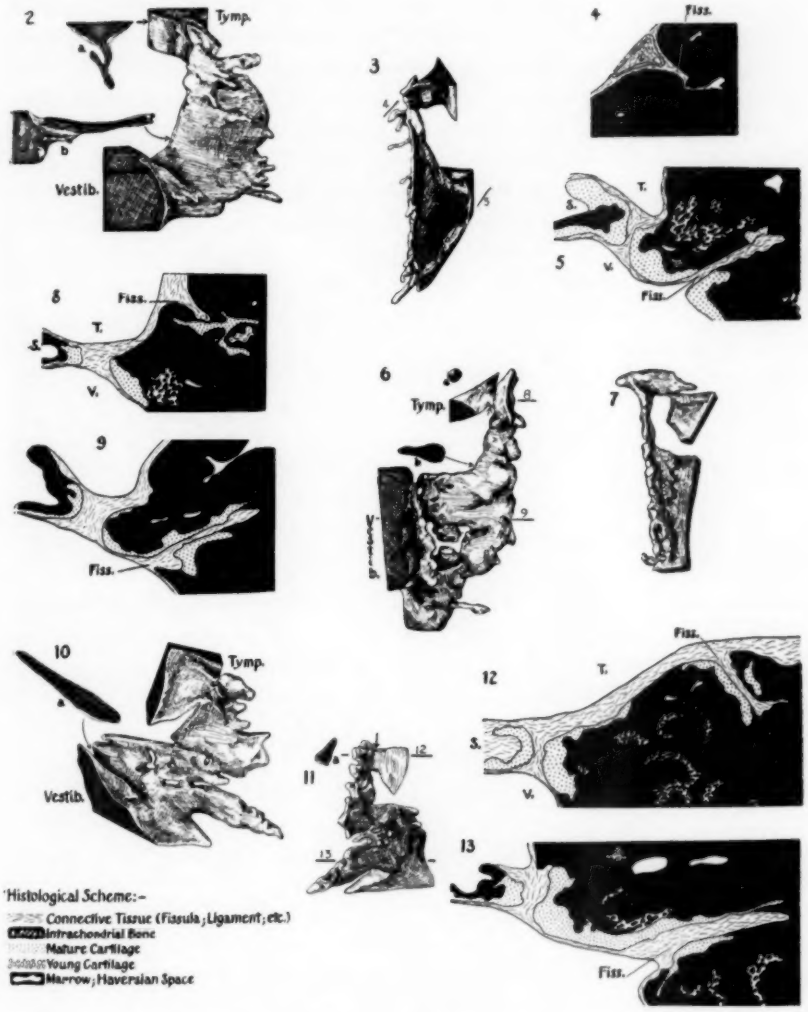
Such also is the shape assumed by the fissula of an adult, 18 years of age (fig. 1), described in 1933 as being typical.[7] In this specimen the fissula begins as a medially directed fault in the bone from the floor of a sulcus which, on the labyrinthine wall of the tympanic cavity, marks the posterior limit of the promontory. After traversing the bone in a curving manner, the fissure ends inferiorly in a larger orifice on the vestibule in front of the anterior part of the base of the stapes. The fissure in this specimen is cleftlike and narrowed mediolaterally; being curvilinear in outline (fig. 3), the anterior limit extends forward toward

7. Anson, B. J., and Wilson, J. G.: The Fissula Ante Fenestram in an Adult Human Ear, Anat. Rec. **56**:383 (July 25) 1933.

the cochlea beyond each of its extremities. The tympanic extremity is longer than it is wide and extends inward (fig. 2), lying in a coronal plane. Then it turns through an angle of almost 90 degrees into a sagittal plane. This change in plane is assumed (figs. 2*a* and *b*) before it passes downward—first forward, next backward—to a vestibular extremity situated near the anterior margin of the vestibular window. Above, the fissula is constricted into an exceedingly slender orifice (fig. 4) and its margins are bone, the original cartilage here having been entirely replaced. Midway in its course the fissular connective tissue is surrounded by modified cartilage (interglobular spaces, or intrachondrial bone). Below, the vestibular orifice is somewhat more ample (fig. 5) and its entire margin on the vestibule is cartilaginous. This cartilage extends medialward a short distance along the wall of the vestibule and lateralward as well, where it is continuous with the cartilage which lines the vestibular window. It is the persistent cartilage of the embryonic fissula, the formation of which has already been described.

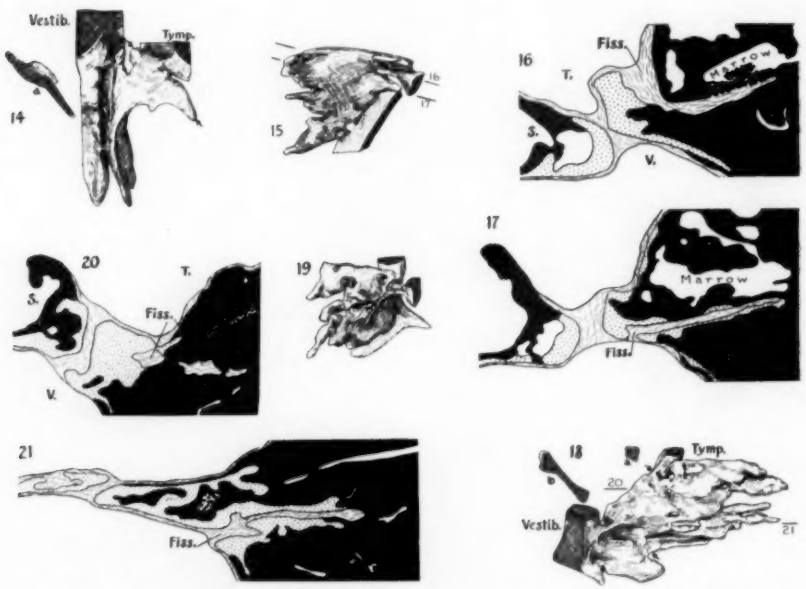
In older persons also the fissula frequently retains a simple form, as in a specimen from a subject aged 57. Here the tympanic extremity begins at the bottom of a funnel-shaped depression which is somewhat flattened dorsoventrally (fig. 6). It is on a horizontal level with the base of the stapes, while in the preceding specimen the tympanic orifice lies in a more cranial position. The orifice itself is like a short cylindric tube (fig. 6*a*) which joins a horizontally placed alar expansion (fig. 7). The main body of the fissula then extends downward as a more capacious cleft than that seen in the preceding specimen (compare figs. 6*b* and 2*b*). Without extending far anteriorly beyond the position of its tympanic extremity, the fissula enlarges abruptly at the junction of its upper and middle thirds to end in the vestibule as an elongate slit, or, rather, in three closely adjacent portions, the upper one being small, the two lower ones relatively large. Medial to the vestibular orifice is a subsidiary outpocketing of the periotic connective tissue into the bone. This outpouching (fig. 6) is not directly associated with the fissula but may be the remnant of a once wider orifice. The occurrence of such an offset vestibular extremity is not uncommon. As seen in sections the tympanic end (fig. 8) has near it some small persistent masses of cartilage and the vestibular end (fig. 9) is almost completely surrounded by cartilage.

In a specimen from a child 2½ years of age, the outline of the fissula is again roughly C-shaped (fig. 10). It extends toward the cochlea in two chief portions, the lower of which is inclined downward into the plane of the scala. The tympanic opening (fig. 11*a*) is somewhat larger than that in the preceding specimen, and the lower or vestibular orifice (fig. 10*a*) is again elongate, its length being five times



Figs. 2 to 13.—Models and sections of fissulae; × 21. The models include the space of the fissulae, but the sections include also the surrounding structures. The fissulae contain connective tissue (purulent exudate in figure 4). Figures 2 to 5 are for an adult aged 18; figures 6 to 9, for an adult aged 57; figures 10 to 13, for a child aged 2½ years. Figures 2, 6 and 10 represent medial views of models; figures 3 and 7 are anterior views; figure 11 is a lateral view. In these and in succeeding drawings the level and the plane of the auxiliary drawings of the models (*a*, *b*, etc.) are indicated by arrows; the level and the plane of the histologic drawings (*4*, *5*, *8*, *9*, etc.), by heavy lines numbered to correspond with the section.[5] *Fiss.* indicates fissula ante fenestram; *T.* or *Tymp.*, tympanic cavity; *V.* or *Vestib.*, vestibule.

as great as that of the tympanic orifice. The orifice is single and uninterrupted by bony bridges at its point of continuity with the vestibule and the scala vestibuli. The lower half of the body of the fissula (fig. 10), however, is crossed at several points by such bridges. The long axes of the two orifices are almost at right angles to each other (figs. 12 and 13). Both orifices are surrounded by cartilage, and the bone in the general territory of the fissula is largely of the intrachondrial type (fig. 66), a condition regularly obtaining in the young. The absence of blood vessels within the fissular tissue is also a regular feature.



Figs. 14 to 21.—Models and sections of fissulae, showing connective tissue; × 21. Figures 14 to 17 are for an infant aged 5 weeks; figures 18 to 21, for a child aged 3 years. Figure 14 represents a craniomedial view; figures 15 to 19 are lateral views; figure 18 is a medial view. Figure 14*a* shows a vertical cut made in the direction shown by the arrows.

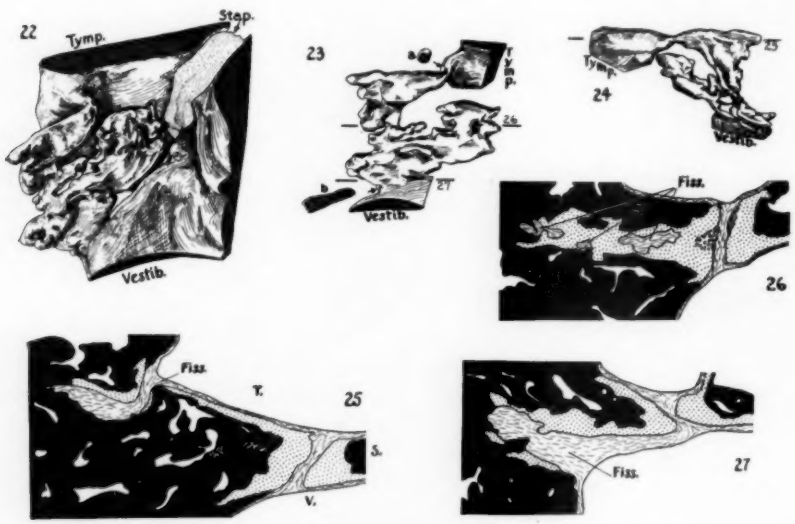
In a specimen from an infant of 5 weeks the fissula is greatly narrowed and of unusual form: Beginning in a tympanic extremity, elongate in the horizontal plane (fig. 14), the fissula quickly widens; then, continuing as a narrow cleft (fig. 14*a*), it turns gradually through an angle of 90 degrees, and descends vertically to its vestibular termination. The vertical portion of the body (fig. 15) is even larger than the horizontally placed portion. Here, in contrast to the arrangement noted in the three preceding examples, the two extremities are in close proximity, only three sections intervening between the lower limit

of the tympanic orifice and the upper limit of the vestibular orifice; both are near the vestibular window (figs. 16 and 17), lying within bays which are separated from the margin of the window merely by jutting ledges of the cartilage which lines the fenestra. This is the only specimen encountered in which the tympanic extremity is wider at any level than the vestibular extremity. At the upper, or tympanic, orifice (fig. 16) the cartilage does not extend into the depths of the fissula, but it does do so at the vestibular orifice (fig. 17). Throughout its course the fissula is adjacent to large marrow spaces, yet it does not at any point communicate with them.

In another specimen, from a child 3 years of age, the fissula resembles that seen in the child of 2½ years. Again the length of the roundish tympanic extremity is one-fifth that of the slitlike vestibular extremity (figs. 18*a* and *b*). The fissula, unlike that in the 18 year old and in the 57 year old adult, is short in the craniocaudal direction (figs. 18 and 19). As is usual, the fissula projects anteriorly toward the cochlea, but in this instance it does so in two main portions, the lower of which is bifid. Both extremities are situated near the vestibular window. The wall of the tympanic orifice is formed by mature cartilage that is continuous with that lining the window. This cartilage follows the fissula inward for a distance (fig. 20) and at the opposite extremity lines the vestibular orifice (fig. 21).

In a specimen from a full term fetus the portions of the fissula adjoining the extremities are large (figs. 22 to 24) and are joined by a small neck. The upper portion extends toward the cochlea, but the lower and larger one projects, not toward the cochlea, but in the direction of the vestibular window, with which it almost communicates. Such extension to the stapedial joint is a common feature in the embryo and in the fetus, having been reported by Bast[6] in seven specimens of 50, 100, 161, 210, 290, 305 and 370 mm., respectively. Beginning at the bottom of a depression in a small cylindric tympanic end (fig. 23*a*) the fissula quickly widens into a cone-shaped space. Through a small communication this space is continuous with a larger one, inferiorly placed and of an irregularly quadrilateral outline. At one point the walls meet (represented by a fenestration in the negative model). The inferior part of the fissula joins the scala vestibuli through a compressed oval orifice (fig. 23*b*). The tympanic end, in part surrounded by cartilage (fig. 25), turns at a right angle in its course, and at its midpoint the fissular connective tissue is embedded in cartilage which is continuous with that of the vestibular window and by a thin lamina of which it is separated from the stapedial ligament (fig. 26). Similarly at the vestibular orifice cartilage forms the wall (fig. 27). The latter extremity is considerably larger than the tympanic (figs. 23*a* and *b*), a relationship which commonly obtains.

In a specimen from a child 5 years of age, the fissula is of simpler form and passes in a rather direct course from a small tympanic extremity to a larger expanded vestibular extremity (fig. 28). The tympanic extremity, although small and tubular (fig. 28*a*), begins at the bottom of a considerable pit which includes the space just anterior to the vestibular window (fig. 30). The necklike extremity extends medialward (fig. 29) to join a larger body (figs. 28*a* and *b*) with pronged extensions above and lobulate ones below. The body then passes downward and backward, its long axis having turned through an angle of 90 degrees in relation to that of the upper or tympanic extremity. The vestibular orific is relatively capacious (fig. 28*c*). Both extremities are

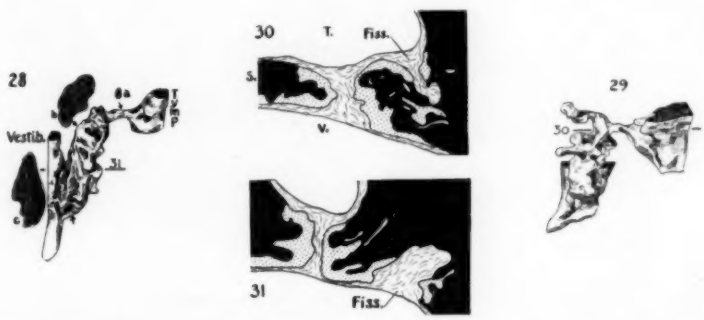


Figs. 22 to 27.—Models and sections of a fissula from a fetus at term, showing connective tissue; × 21. Figure 22 represents an anteromedial view; figure 23, a medial view; figure 24, a lateral view. *Stap.* indicates stapedial ligament (stippled in figure 22).

situated near the margin of the vestibular window (figs. 30 and 31) and are partially lined by cartilage which is continuous with that of the fenestra.

It is evident that at least a part of the wall which encloses the space of the fissula is regularly composed of cartilage, persistent within the otic capsule from the stage at which the capsule was wholly cartilaginous. Cartilage, however, in some specimens is not confined to the walls as a mere tubular investment for the connective tissue but actually occurs as a mass which is continuous with the fibrous tissue or wholly replaces it. It is not altogether surprising that such histologic variations occur

in specimens from young persons, or that they are accompanied in these active areas by striking aberrations in the form and in the size of the fissula. In a full term fetus some of these unusual characters are found. The lower portion of the fissula is similar in several respects to fissulae that are commonly encountered, but an upper extension therefrom is aberrant. The lower portion ends conventionally above and below in orifices that are filled with connective tissue. The small round upper extremity (fig. 32*a*) extends inward a short distance, meeting the body of the fissula at a right angle. The flattened body passes through a constricted portion into an inferior expansion (fig. 33), its general form suggesting that of the fissula in the other full term fetus. The vestibular communication is unusually small (fig. 33*a*). Both extremities are removed from the vestibular window by more than the usual distance (figs. 34 and 35), yet the fenestral rim of cartilage is carried



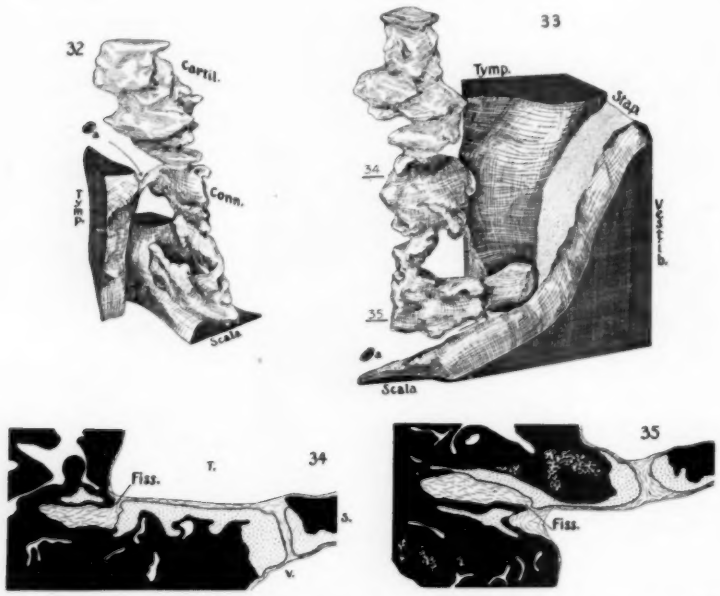
Figs. 28 to 31.—Models and sections of a fissula from a child 5 years of age, showing connective tissue; × 21. Figure 28 represents a medial view; figure 29, an anterior view.

anteriorly to invade the orifices. As thus far described, the fissula in this specimen, save for the reduced lower orifice, is not strikingly exceptional, but it contributes a feature not occurring in the other specimens herein described [8] in possessing a superior prolongation (fig. 32) two-thirds as long as the fissula proper and composed entirely of immature cartilage. This mass surmounts the connective tissue at a level immediately above that of the tympanic extremity, where the two parts are joined through a zone of perichondrial tissue (stippled, fig. 32).

In a specimen from a 3 year old child, cartilage occurs within the area between the two extremities, whereas in the preceding specimen it

8. In discussing our models recently Dr. T. H. Bast stated that the occurrence of cartilage in the fissular territory is a condition encountered in a high percentage of young persons; the less frequent occurrence of such cartilage in older persons and the problem which these statistical facts present will, we understand, be treated by Dr. Bast in a paper now in preparation.

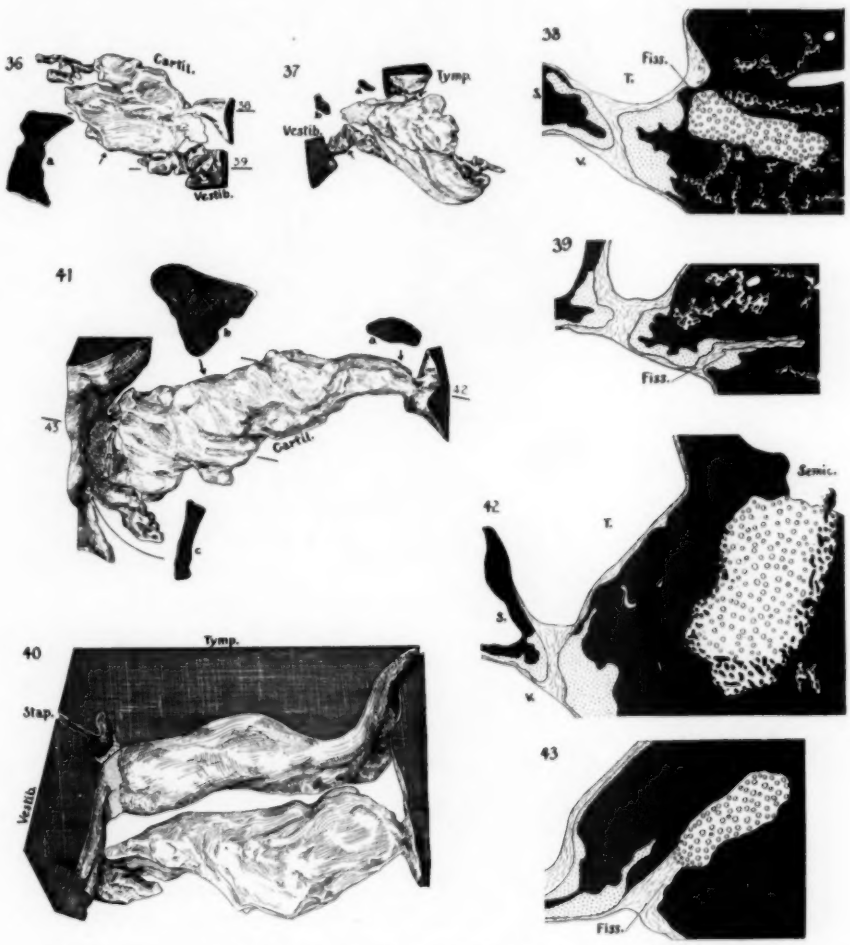
occurs only as an appendage beyond the limit of the superior extremity. This tissue becomes continuous with the perichondrial tissue and with the connective tissue. The small tympanic orifice of the fissula lies at the bottom of a conical recess (fig. 36) which contains submucosal connective tissue. The vestibular orifice is somewhat larger. Both orifices are flattened when seen in cross-section (figs. 37*a* and *b*) and are situated near the vestibular window (figs. 38 and 39). Through a short neck of perichondrium, as intermediate tissue, the tympanic connective tissue merges into cartilage (figs. 36 and 37), which occupies the customary position of the cleft in the bone.



Figs. 32 to 35.—Models and sections of a fissula from a fetus at term, showing connective tissue and cartilage; × 21. Figure 32 represents an anterior view; figure 33, a medial view. *Cartil.* indicates cartilage; *Conn.*, connective tissue; *Scala*, scala vestibuli.

At the cochlear extremity a little of the original space remains (having the form of a bifid prong in the model). The mass of cartilage is relatively bulky, being almost one-third as wide as it is high (fig. 36*a*). Inferiorly, a space (with its contained connective tissue) again occurs and constitutes a vestibular extremity. At the extremities (figs. 38 and 39) cartilage bounds the orifices, but here, as in all other similar specimens, it is readily distinguishable from the newly formed cartilage which occupies the fissular territory (see description of specimen from a 2 year old child on page 315.

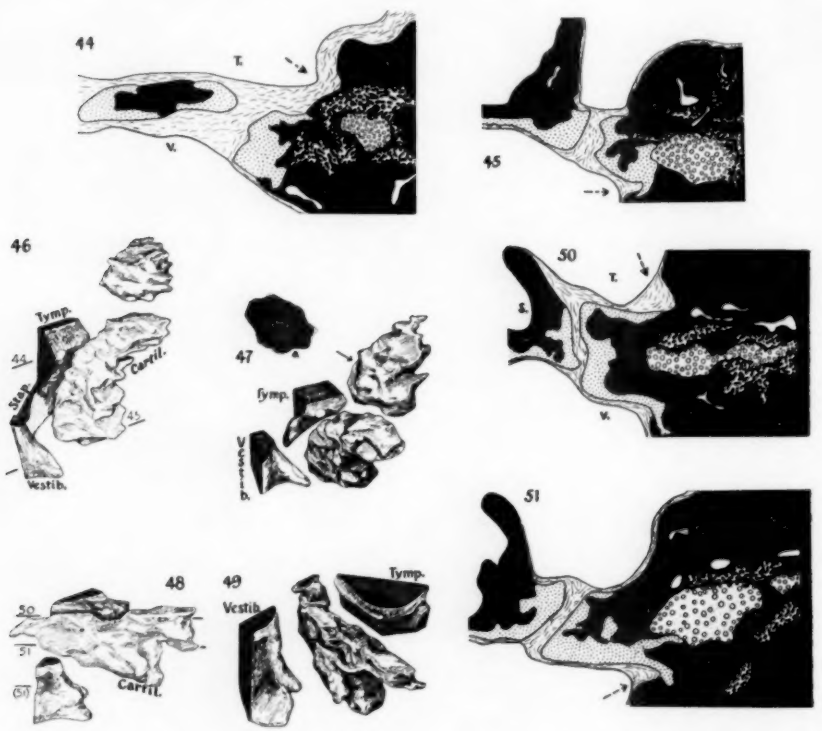
In a specimen from a child 5 years old, the fissular area is almost wholly occupied by cartilage of the immature type, only the vestibular portion persisting as connective tissue. Whereas the common form of fissula is that of a structure bent on itself so that its body stretches away



Figs. 36 to 43.—Models and sections of fissulae from a child aged 3 years (figs. 36 to 39) and a child aged 5 years (figs. 40 to 43), showing connective tissue and cartilage; × 21. Figure 36 represents a lateral view; figure 37, a cranial and anteromedial view; figure 40, a craniomedial view; figure 41, a medial view. In figure 41 the connective tissue lies to the left of the dotted line; the cartilage, to the right of it. In figure 42 the tensor tympani muscle has been omitted. *Semic.* indicates the semicanal for the tensor tympani muscle.

into a position well anterior to that of either of its ends, in this specimen the course is almost straight; it passes to the vestibule from a tympanic

extremity on the wall of the semicanal for the tensor tympani muscle (fig. 40), a relatively great distance from the vestibular window. The fissular area here is situated in the thicker portion of the capsular wall and is to be contrasted with the fissula of the infant (figs. 16 and 17), in which the course is through the narrowed bone near the fenestra. The fissula of this 5 year old child is of proportionately large dimensions throughout, not only near the midportion (fig. 41*b*) but even at the tympanic end (fig. 41*a*). It is broad near the tympanic end



Figs. 44 to 51.—Models and sections of fissulae from a child aged 2 years (figs. 44 to 47) and a child aged 6 years (figs. 48 to 51), showing cartilage; × 21. Figures 46 and 48 represent medial views; figures 47 and 49, cranio-anterior views. The arrows in figures 44, 45, 50 and 51 point to the presumptive sites of the orifices.

(fig. 40) and deep near the opposite end (fig. 41; compare *a* and *c*). Cartilage occupies almost all of what was once fissular space within the bone, and at the tympanic extremity the cartilage abuts on the semicanal (figs. 42 and 67). It does not reach the vestibular orifice, however, and connective tissue still occupies that space (fig. 43).

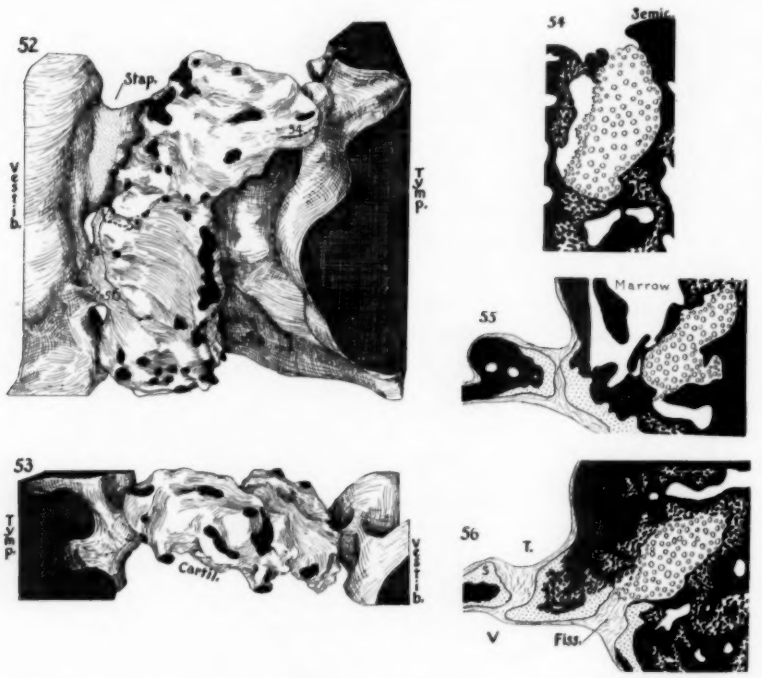
In a specimen from a 2 year old child, no connective tissue remains in the fissular area, the fissula being represented by two discontinuous masses of cartilage, no portion of which reaches the surface of the bone. The lower mass (fig. 46) occupies a position comparable to that held by the connective tissue in the fissula of the fetus (fig. 32); the upper mass occupies a place which is rather similar to that held by the cartilage in the fetus, but instead of ascending it follows the more common plan of extending anteriorly toward the cochlea (fig. 47). Although no true connection exists between the fissular tissue and the periosteum on either surface of the bone, an indentation is present at the expected site on both the tympanic and the vestibular surface (figs. 44 and 45 at arrows). The indentation on the vestibular surface is the more evident, and its cartilage is in contact with, yet histologically distinct from, that which extends inward from the vestibular window (fig. 45). The same varieties of cartilage are observed deeper in the bone; the mature type here partially encapsulates the younger in a manner similar to that in which it forms the wall about the fissular connective tissue in specimens from older persons (figs. 5 and 13). Through the major portion of its course the fissular cartilage lies in a collection of calcified cartilage or intrachondrial bone (fig. 44), by which assumedly it would in time have been at least partially replaced.[9]

In a specimen from an older child (6 years of age) the fissular area is again occupied by cartilage, but in a single undivided mass which, like that in the preceding specimen, is prolonged superiorly toward the cochlea (figs. 48 and 49). Although no part of the mass terminates as an extremity on the periosteum, an indenture filled with connective tissue is present on each surface of the bone, marking the presumptive site of the original orifice. The tympanic pit (fig. 50) is near the vestibular window and like the vestibular pit (fig. 51) lies within the zone of the fenestral shell of cartilage, by which it is lined.

In a specimen from a 2 year old child (fig. 52) the occurrence of precociously developed immature cartilage in the fissular territory makes clear the manner in which such tissue gradually displaces first the connective tissue of the fissula and then the adjacent marrow channels to produce a chondral nodule within the bone where the fissular

9. In examining our sections Dr. T. H. Bast expressed the opinion that wherever the developing invasive cartilage encountered thin plates of endosteal bone (as in fig. 69, left side), the latter would give way, permitting ingress of the cartilage into the marrow spaces and thus its spread beyond the confines of the original fissular area. Basing an opinion on his study (1930) of intrachondrial bone (variously called calcified cartilage, interglobular spaces and cartilage islands) and the method of its formation in the fetal otic capsule, Dr. Bast further stated that he regarded the vacuolated condition of the cartilage in the specimen shown in figure 68 as preparatory to the invasion of this tissue by osteogenetic buds and its resultant conversion into intrachondrial bone.

channel lay earlier. In this specimen the cartilage mass passes directly between the extremities on the tympanic and the vestibular wall (figs. 52 and 53), descending as it passes medialward. It bears low processes marking the points at which its tissue projects into the abundant surrounding marrow. At the tympanic orifice the new cartilage presses beyond the free surface of the tympanic wall into the semicanal of the tensor tympani muscle (fig. 54); at intermediate points it displaces marrow (fig. 55) and at the vestibular extremity it pushes toward the mouth of the orifice (fig. 56), in which, however, some of the



Figs. 52 to 56.—Models and sections of a fissular area from a child aged 2 years, showing cartilage, connective tissue and marrow; × 21. Figure 52 represents an anterior view; figure 53, a cranial view. In figures 52 and 53 the blackened portions represent areas of contact of the cartilage with marrow. In figure 54 the tensor tympani muscle has been omitted.

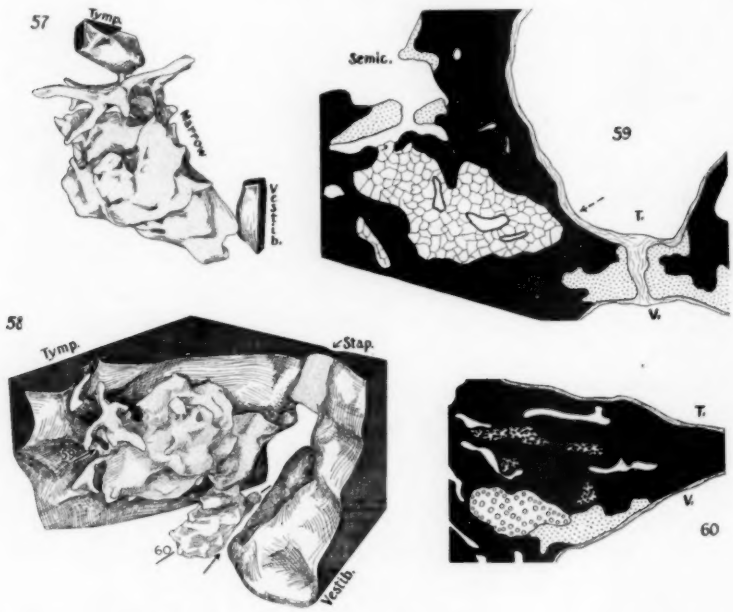
connective tissue still remains. At each extremity of the mass there is a gradual transition from perichondrial connective tissue into newly formed cartilage. Between the latter and the surrounding intrachondrial bone, however, a sharp line of demarcation indicates a marked difference in age; the intrachondrial bone, on the contrary, passes gradually into the older cartilage of the fissular wall, of which it is a derivative.

there being complete transitional stages between the two. At the vestibular extremity these several tissues may be seen (fig. 68). The persisting connective tissue of the fissula at the bottom of the vestibular pit becomes a transitional perichondrial tissue capping the plug of newly formed cartilage. The three tissues become continuous by insensible gradations. The new cartilage, with its investment of perichondrium, is encroaching on the fissular orifice, growing toward the vestibule as a tonguelike projection. A similar series of transitional stages is found at the numerous points at which the new cartilage encounters marrow spaces and forms bulging prominences into them, as indicated by the arrow in figure 68. Of a different order is the cartilage which lines the fissular orifice and against which the growing cartilage lies. This is mature tissue which has been present in this locality from the stage of embryonic formation.[10] It passes imperceptibly into the intrachondrial bone which in the deeper portions of the fissula intervenes between the mass of young cartilage and the bone wherever the cartilage is not adjacent to marrow. At no point does the mature cartilage or the intrachondrial bone pass by an intermediate transitional tissue into the young cartilage which occupies the fissula. Evidently, then, the young cartilage is of relatively recent formation and is spreading peripherally, through the activity of its perichondrium, toward the orifice of the fissula and into the surrounding marrow spaces. At the tympanic extremity (fig. 69) the new cartilage not only has spread beyond the mouth of the orifice into the semicanal but has almost completely obliterated the adjoining marrow spaces (fig. 69 at arrow), reaching the intrachondrial bone, from which it is structurally distinct. The intrachondrial bone, however, is continuous with the surrounding bone. Whether or not, in such a case, the cartilage would ultimately be transformed by invasion into intrachondrial bone is a question which cannot be answered from an examination of our material.[9]

The original connective tissue of the fissula in some specimens apparently acquires secondary communications with neighboring marrow spaces. We regard the preceding specimen as one in which marrow, having displaced ordinary connective tissue, was in turn largely supplanted by young cartilage. In some specimens of temporal bone,

10. In the mature cartilage (fig. 68) the ground substance is definite, forming a dark-staining matrix in which the small cartilage cells lie in lacunae the margins of which are sharply outlined. The lacunae are of a compressed oval shape, with the long axes coinciding with the free surface of the cartilage. Within the newly formed immature cartilage the lacunae are large, are not arranged in definite pattern and lie within a matrix which is light-staining and continuous with the more cellular perichondrium from which it was derived by appositional growth. The new cartilage resembles structurally a hyaline chondroma.

marrow in preponderant amount still remains in the fissular area, associated with a relatively small mass of young cartilage. Thus, in one adult (age unrecorded) marrow tissue in a state of fatty degeneration occupies the upper portion of the usual fissular territory (figs. 57 and 58) and, like the cartilage in the 5 year old child (fig. 42), terminates in the middle ear by opening on the semicanal for the tensor muscle. The lower portion, which is perfectly continuous with the upper, is composed of young cartilage. The upper portion of the marrow projects into the surrounding bone by pronglike extensions, and it is through

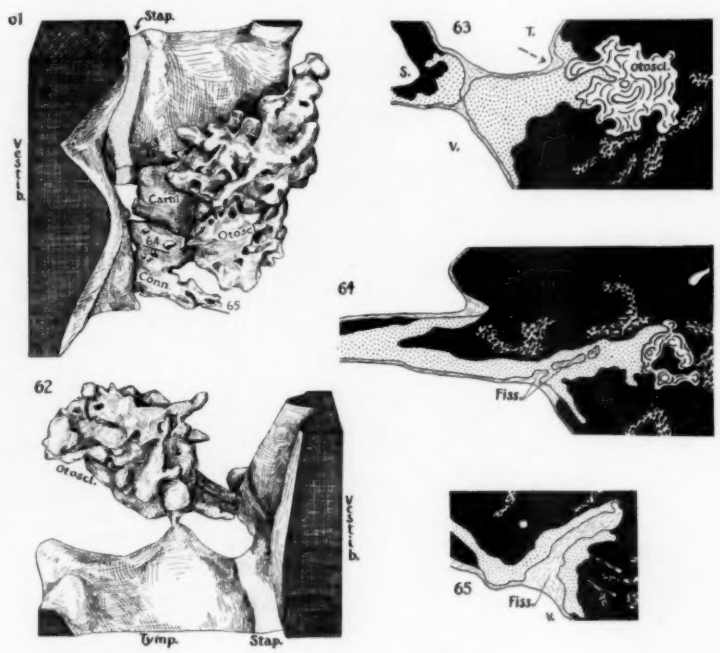


Figs. 57 to 60.—Models and sections of a fissular area from an adult whose age was unrecorded, showing cartilage and marrow; × 21. Figure 57 represents a cranial view; figure 58, a cranial and anteromedial view. In figure 58 the arrow at the left not only marks the level of the section in figure 59 but points to the tympanic orifice of the fissula into the semicanal as seen in figure 59; the arrow at the lower right in figure 58 points to the site of the mature cartilage at the vestibular extremity, as seen in figure 60.

one of these that the mass communicates with the connective tissue of the semicanal (fig. 58). The tympanic orifice is small (fig. 58, arrow at left), but the marrow cavity with which it communicates is capacious, extending toward the vestibular window and at one point (fig. 59, at arrow, and fig. 70) being separated from the tympanic cavity merely by a thin plate of bone. At the vestibular end (fig. 60) young cartilage

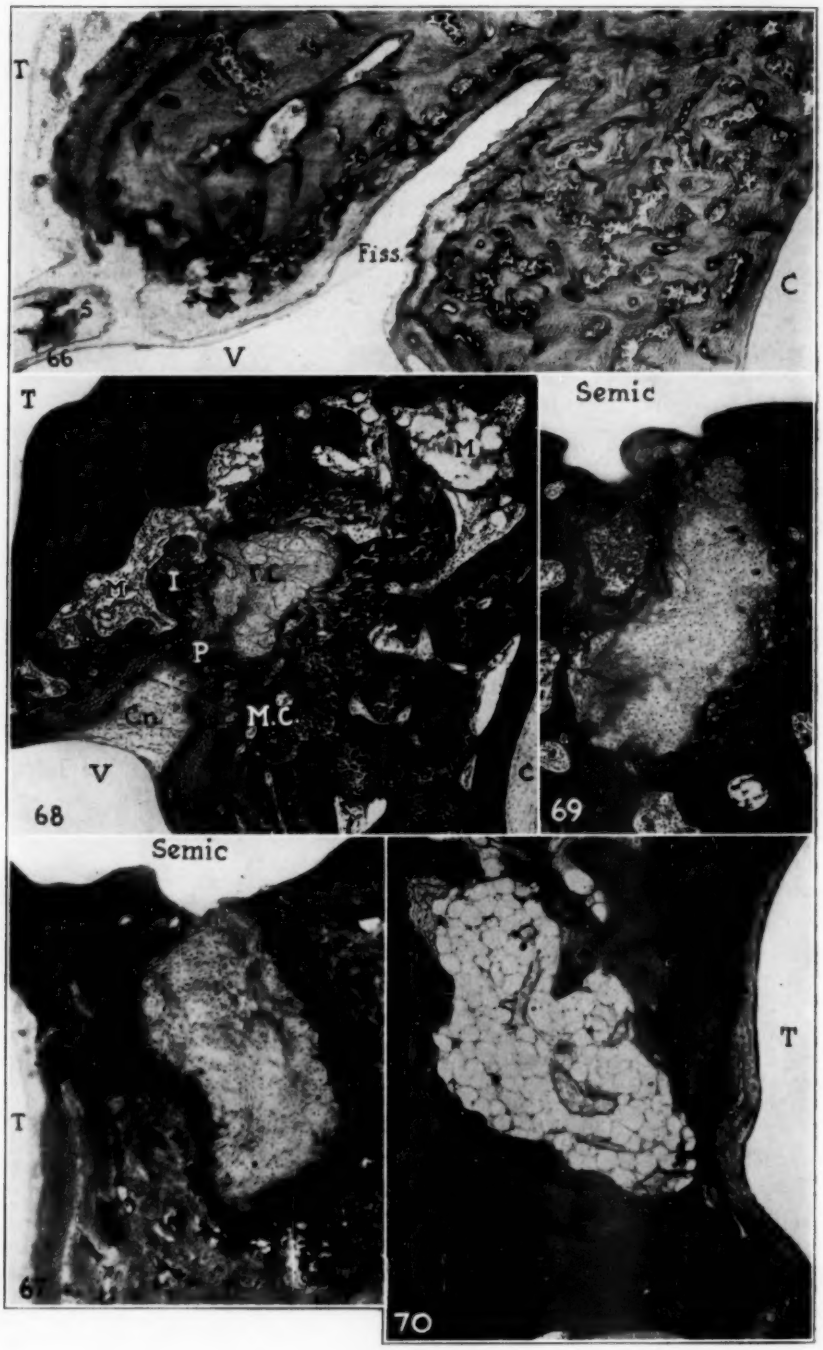
occupies the fissular space. Where one would expect to find a vestibular orifice, mature cartilage, usually present at the orifices, extends into the bone and meets the young cartiiage. This, then, is the third specimen in the group in which an orifice on the vestibule is wanting.

Cartilage and marrow are not the only tissues that occupy the customary position of the fissula, for not infrequently sclerotic bone occurs in this area, partially or wholly obliterating the fissular connective tissue. In a specimen from an adult, 18 years old, the fissular area is remarkable for the manner in which it terminates conventionally below but



Figs 61 to 65.—Models and sections of a fistular area from an adult, aged 18, showing connective tissue, cartilage and sclerotic bone (the sclerotic bone shown as whorled in figs. 63 and 64); × 21. Figure 61 represents a medial view; figure 62, a cranial view. *Otoscl.* indicates otosclerosis, or sclerotic bone.

widens above into a relatively large nodule of sclerotic bone; between the two portions a cartilaginous strip intervenes. At the inferior extremity (fig. 61) the communication with the vestibule is elongate and somewhat broken by areas of continuity of the two walls (fenestrations in the model); from the lower portion an irregular projection of the space extends toward the cochlea. The fibrous tissue gives way to cartilage, the two tissues merging through a narrow band of perichondrium. The preponderant part of the entire mass is composed of a vascular, spongy



Figs. 66 to 70

EXPLANATION OF FIGURES 66 TO 70

Figs. 66 to 70.—Photomicrographs of horizontal sections through the fissular area in three specimens; × 42. Figures 67 and 69 have been retouched, and the contents of the semicanal were removed to show more clearly the extent of the fissular cartilage. *C* indicates cochlea. Figure 66 shows the vestibular orifice of the fissula in a 2½ year old child (same section as depicted in figure 13). The tympanic mucous membrane is edematous. Figure 67 shows the tympanic extremity of the fissular tissue in a 5 year old child (same section as depicted in figure 42). It illustrates the relation of the young cartilage within the fissula to the semicanal of the tensor tympani muscle. Figure 68 shows the vestibular orifice of the fissula in a 2 year old child (section made near that depicted in figure 56). Young cartilage replaces the fissular connective tissue. *Cn.* indicates connective tissue; *I.*, intrachondrial bone; *I.C.*, immature cartilage; *M.*, marrow; *M.C.*, mature cartilage; *P.*, perichondrial tissue. The arrow points to the cartilaginous projection into the marrow space. Figure 69 shows the tympanic extremity of the fissular area from the same specimen (section made near that depicted in figure 54). Young cartilage protrudes beyond the orifice into the semicanal and obliterates the marrow space (at arrow). Figure 70 shows the tympanic extremity of the fissular area of an adult (same section as that depicted in figure 59). The orifice opens on the semicanal; the fissula contains fatty marrow.

bone of the sclerotic type [11] which extends cranialward (fig. 61) and anteriorly toward the cochlea (fig. 62) well beyond its tympanic and vestibular connections. The mass is strikingly irregular, bearing numerous projections and being frequently perforated by enlarged haversian channels. Above, at the tympanic extremity, the sclerotic bone is exposed to the mucous membrane without the intermediation of cartilage or connective tissue. Here the relatively minute termination lies at the bottom of a cone-shaped indenture and is bordered by adult cartilage (fig. 63) which is part of the shell which lines the vestibular window and inferiorly extends along the vestibular wall and into the vestibular opening of the fissula (figs. 64 and 65).

SUMMARY AND CONCLUSIONS

It is well known that the periotic capsule in man regularly contains a remarkable group of connective tissues, varying between the primitive and the advanced types. Thus, within the periosteal layer, primary bone persists, unreplaced by lamellar bone; in the form of scattered islands, a calcified cartilage is present, the matrix of which has been rendered osseous through the activity of osteogenetic buds.[12] Connective tissue proper occurs as a fibrous stripe within the lateral wall of the osseous vestibule and just in front of the vestibular window, where it occupies a minute channel known as the fissula ante fenestram. Lastly, unaltered hyaline cartilage is found, restricted in its distribution to the orifices of the fissula on the tympanic cavity and the vestibule and to the nearby vestibular window.

Our study shows that this fissula or channel not only assumes various forms but may, especially in the young, be secondarily increased in extent through communications with neighboring marrow spaces or reduced in size through the encroachment of new cartilage or of sclerotic bone.

The fissula is not infrequently incomplete, lacking one or both orifices. The extremities of the fissula may vary in relation to dependable landmarks: the tympanic orifice may open, not near the vestibular window, but into the semicanal for the tensor tympani muscle; the vestibular

11. The macroscopic nodule possesses the several features commonly regarded as characteristic of otitic sclerosis. It stains deeply with eosin; it contains large, thin-walled blood vessels in capacious, ramifying channels which give to it a spongious appearance; the channels are enclosed by definitely lamellar bone. Assumedly, if further spread, the new bone would obliterate the normal fissular tissue and even the surrounding bone—a change accomplished in a case described by Bast and by Wilson and Anson in 1933 (Arch Otolaryng. **18**:291 [Sept.] 1933).

12. Bast, T. H.: Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. **21**:53 (June) 1930.

orifice may open, not into the vestibule proper, but into the scala. Yet, in spite of the occurrence of such variation, so far as observed, the tympanic orifice has been situated invariably in a more cranial position than the vestibular, and with one exception it was found to be the smaller of the two openings. The vestibular orifice may be of relatively reduced size. It is sometimes crossed by bridges of bone, suggesting that, once it is established in the embryonic temporal bone, both its shape and its size may be altered by the subsequent growth of osseous tissue. There is ample evidence too that normal bone is not the only tissue that remains productive in the fissular area, since, with surprising frequency in young subjects, immature cartilage is found in what undoubtedly was fissular space. We have found several examples of such encroachment: one in which the newly formed nodule of cartilage occurs as an appendage to an otherwise typical fissula, beyond the limit of its superior extremity; three in which the cartilage almost completely fills the original fissular space, only the vestibular portion in each persisting as a space containing fibrous tissue, and two in which cartilage has obliterated all of the space, lying embedded in the bone without tympanic or vestibular termination. In one of these six specimens the cartilage is still in an active state, possessing perichondrial extensions into the orifices of the fissula. Occasionally, sclerotic bone in rapid development obliterates part or all of the fissular space. Whether or not such bone must genetically succeed the young cartilage is a question to which our observations at present give no answer. It may be said, however, that the frequent occurrence of otosclerosis within the fissular territory leads one to suspect that a definite histologic succession occurs in this histologically mobile area. Our further study will be concerned in part with this problem of differentiation of connective tissues within bone commonly considered to be virtually unchanging.

We can assign no function to the fissula ante fenestram. We do not agree with those who regard it as a synchondrosis: The relatively small size of the fissure, its location as a minute channel within one bone rather than as a union between bones, its histologic variability and its fortuitous occurrence as a complete structure all militate, we believe, against such an interpretation. Possibly the study now in progress, which deals especially with older specimens, will bring to light some function subserved by the fissula.

LOCAL TUMOR-LIKE DEPOSITS OF AMYLOID IN THE LARYNX

REPORT OF A CASE WITH A REVIEW OF THE LITERATURE

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The occurrence of isolated tumor-like deposits of amyloid in such areas as the upper air passages may have a variable significance depending on associated pathologic conditions in the body as well as on the presence or absence of a basic etiologic factor. The following classification into four groups is based on an analysis of published reports of cases in the literature:

1. Local amyloid deposits occurring secondarily within neoplasms or areas of chronic inflammation. This type should be distinguished from primary local amyloid tumors which have no obvious basis in preexisting changes of the tissue. Secondary amyloid tumors are well known and have been reported frequently, especially in fibromas and sarcomas (Martuscelli). Peroni described amyloid infiltration also in inflammatory polypi of the larynx and the ear.

2. Local tumor-like amyloid deposits which are part of a generalized amyloidosis of the usual type involving liver, spleen, kidneys and suprarenal glands. Such local deposits may involve the upper respiratory tract. Exceptionally a local deposit may precede the generalized amyloidosis. Beckert reported a case of amyloid tumor of the upper air passages in which generalized amyloidosis did not develop until several years later. He believed that there occurred a gradual transition from local to general amyloidosis.

3. Local amyloid tumors which are part of a generalized amyloidosis of atypical character. In this form the viscera involved in the usual type of generalized amyloid disease are spared; instead the infiltration is peculiarly restricted to such structures as the striated and smooth muscles, lungs, heart and skin. Localized amyloid tumor-like deposits in the upper air passages may occur in such instances. Lubarsch described three cases of atypical amyloidosis and postulated several criteria for the classification of this type: (1) almost complete exemption of the organs characteristically infiltrated by amyloid in the usual type of

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From the Rhinolaryngologic Service and the Laboratories of the Mount Sinai Hospital.

case; (2) deposition of amyloid in organs usually spared; (3) a tendency to the formation of nodular depositions of amyloid; (4) atypical reaction of the amyloid to the usual specific stain, and (5) the absence of a demonstrable underlying cause (e. g., chronic suppuration).

Recently Gerstel reported a case of diffuse amyloid tumor of the tongue with similar tumors in the muscle, the duodenum and the skin. These tumors had been clinically diagnosed as metastatic new growths.

4. Idiopathic or primary tumor-like amyloid deposits. In the cases that fall into this category there are single or multiple amyloid deposits confined to a single anatomic region and unaccompanied by amyloid infiltrations elsewhere in the body. There is no definite etiologic factor demonstrable. Evidences of preexisting inflammation or new growth are absent. These formations have been called amyloid tumors to indicate their tumor-like gross appearance, but they are not to be considered true blastomas. The most frequent localizations are the upper air passages and the conjunctiva, and they have been noted also in the bladder and the stomach.

Herxheimer was able to collect reports of twenty-four such cases up to 1903. It was not until Pollak published a comprehensive analysis of idiopathic amyloid deposits in the upper air passages that the lesion was recognized as a distinct entity. After carefully analyzing the previously reported cases and his own he established the following characteristic picture for local amyloid tumors: (1) multiplicity of lesions in the involved organ, especially in the larynx; (2) involvement of contiguous organs in a similar manner, e. g., the cheek, tongue, larynx and trachea; (3) transparent, waxy appearance of the tumors, and (4) absence of ulceration, glandular involvement and pain.

Although the majority of instances of this regional type of idiopathic amyloidosis conform to these criteria, true cases may occur in which certain characteristics are lacking. The case here reported is an example of the type in which involvement of adjacent organs could not be demonstrated.

REPORT OF CASE

History.—M. L., an Italian girl 19 years old, was admitted to the rhinolaryngologic service of the Mount Sinai Hospital on April 24, 1932. She had enjoyed good health all her life and said she had not had a previous serious illness. There was no history of frequent colds or cough. Three and one-half years prior to admission the patient noted the gradual onset of hoarseness which became progressively worse. There was no antecedent infection of the upper respiratory tract or any known immediate or related cause. The patient had been a member of a church choir for several years. Of late she had experienced slight stridor on exertion. There was no loss of weight.

Examination.—Physical examination revealed a well developed girl in apparent good health, who spoke in a whisper. Except for her local laryngologic condition the physical examination gave negative results. The trachea was in the midline and freely movable. There was no abnormal enlargement of lymph nodes

in the neck or elsewhere. The heart and lungs were normal. The systolic blood pressure was 136 and the diastolic 72. Examination of the urine revealed no abnormalities. The Wassermann reaction of the blood was negative on two examinations. A roentgen examination of the neck revealed a globular mass about 2.5 cm. in diameter apparently originating in the vestibule of the larynx and impinging on its lumen anteriorly.

Laryngologic examination revealed prominent lateral pharyngeal folds. The tonsillar fossae were clear. Resting on the right false vocal cord and ventricle was a well circumscribed nodular grayish mass about 2 cm. in diameter, apparently attached to the right laryngeal wall and aryepiglottic fold. A small nipple-like protrusion could be seen on the mesial surface of this mass. In addition, there were several small prominences along the false cord anteriorly extending into the anterior commissure. Since the main tumor encroached considerably on the laryngeal lumen, visualization of the right vocal cord was not possible by indirect laryngoscopy. Ample airway, however, was present for normal respiration. The right arytenoid cartilage moved freely with respiration. The clinical impression was that the tumor was benign.

Operation.—It was not considered feasible to remove the tumor by indirect laryngoscopy because of its rather large size. With the patient under general avertin anesthesia, suspension laryngoscopy was performed. The tumor was seen to arise from the right ventricle and false vocal cord and extend to the anterior part of the left false vocal cord. The mass was grasped by pincettes and pulled forward with a clamp. It felt hard and fibrotic. The base of the tumor appeared to infiltrate the lateral laryngeal wall along a wide area and was not clearly demarcated from the deeper structures. The tumor could not be ablated in toto with a snare but had to be bitten away with a punch forceps. The suspicion of a possible malignant tumor was entertained because of the wide attachment and apparent invasion. Postoperative recovery was uneventful. There was a surprisingly small amount of bleeding. The patient was discharged from the hospital on April 30.

Microscopic Examination.—The section, stained with hematoxylin and eosin, showed homogeneous structureless tissue lined by a layer of pseudostratified columnar epithelium. In some areas hornified squamous epithelium was present. The homogeneous substance was almost acellular and took a faint pink stain with eosin. Focal areas in the vicinity of glandular structures and blood vessels stained more deeply pink. Immediately beneath the epithelium was a slight infiltration of plasma cells, small lymphocytes and occasional polymorphonuclear leukocytes. In one area there was a nest of mucous glands surrounded by concentric layers of hyalinized pinkish-staining material. The provisional diagnosis from the microscopic examination was: hyalinized fibro-adenoma of the larynx.

With aniline dyes the homogeneous substance was identified as amyloid.

Course.—One month following discharge the patient returned for reexamination. Her voice was markedly improved, though not yet normal. A small nodule on the right false vocal cord removed for biopsy showed chronic inflammation on section.

Second Admission.—The patient came under observation again after an interval of sixteen months. Her voice had been definitely benefited by the surgical procedure and had remained improved until four months before, at which time hoarseness recurred. The hoarseness had been progressive. Dysphagia occurred on eating certain foods, and occasionally she had stridor. She was readmitted to the hospital, and complete physical examination again revealed no evidence of generalized

amyloidosis. However, her liver was palpable and there was a question of a possible enlargement of the spleen. Repeated examinations of the urine revealed no abnormality. Reexamination of the neck roentgenologically revealed a mass similar to the original tumor but smaller.

On indirect laryngoscopy the mass was found to involve the left false cord. It resembled the one previously removed from the opposite side, having a mulberry contour and a grayish color. It was 2 cm. in diameter. It had a glazed appearance and was attached to the lateral wall of the larynx by a broad base. A prolongation of the tumor extended to the left aryepiglottic fold and the anterior commissure. There were numerous localized exuberances scattered over the right wall of the larynx at the site of the original tumor. The mass interfered with

Fig. 1.—A laryngoscopic picture of the amyloid tumor on readmission. Recurrent exuberances can be seen on the right side. A massive amyloid deposit on the left false vocal cord extends across the anterior commissure but causes very little laryngeal obstruction.

complete visualization of the larynx and prevented proper approximation of the vocal cords. There was no regional lymphadenopathy.

With the patient under general avertin narcosis supplemented by cocaine anesthesia a suspension laryngoscopy was again performed. The tumor was not well demarcated from surrounding structures and could not be dissected free from its laryngeal bed. With a punch forceps portions of tissue were removed piecemeal until the perichondrium of the thyroid cartilage was reached. The tissue was waxy in appearance and difficult to excise. The base of the lesion was formed by the false vocal cord, thereby sparing the true vocal cords. At this time no attempt was made to remove the recurrent exuberances on the opposite side of the larynx for fear of postoperative laryngeal stenosis.

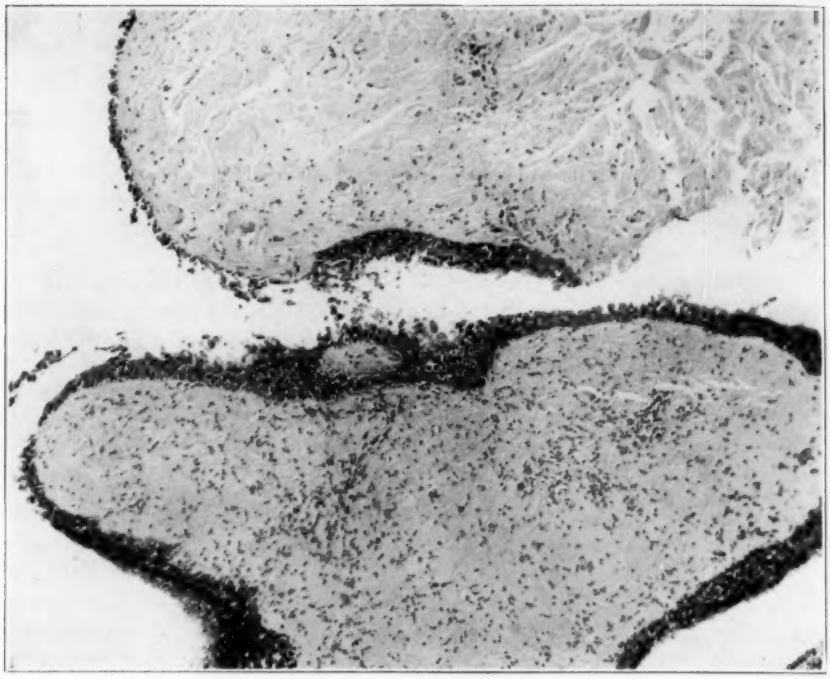

Fig. 2.—A low power photomicrograph (hematoxylin and eosin stain) showing diffuse infiltration by structureless amyloid substance. A mild subepithelial inflammatory reaction is present; the epithelium is intact.

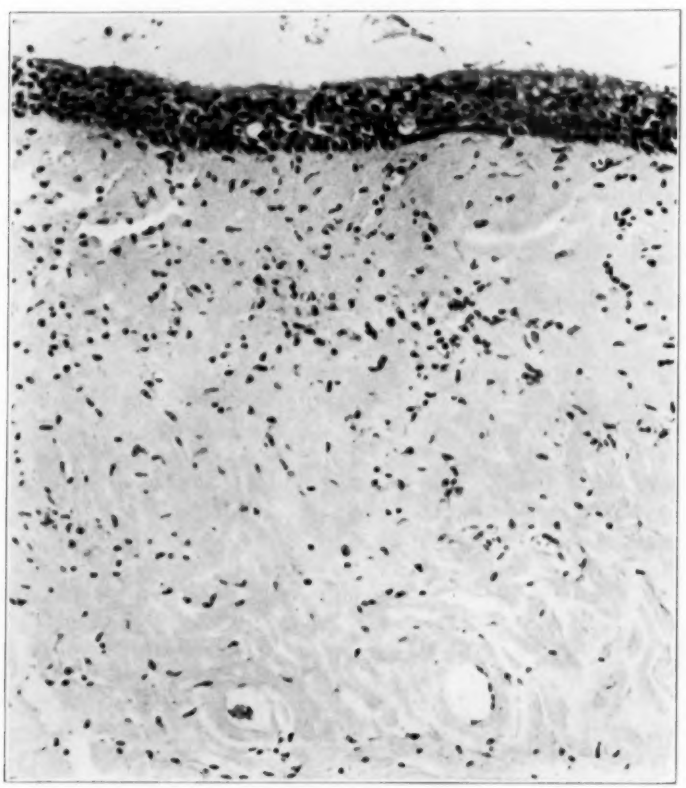

Fig. 3.—A high power photomicrograph, showing the tendency to perivascular deposition of amyloid substance.

Microscopic Examination.—With the exception of the superficial pseudostratified epithelium the entire section, stained with hematoxylin and eosin, took the acidophilic pinkish stain with eosin. The epithelium showed a distinct basement membrane, and occasionally isolated polymorphonuclear leukocytes were seen between the epithelial cells. The stroma consisted of homogeneous tissue which was arranged in the form of irregular islands and strands. Few plasma cells, small lymphocytes and polymorphonuclear leukocytes were found. With the congo red stain the structureless substance was identified as amyloid. In some areas, particularly about the blood vessels, the amyloid deposit seemed concentrated; here the acidophilic staining was intensified. There were occasional nests of mucous glands surrounded by a concentric layer of this homogeneous substance. Several glandular alveoli were dilated, and their epithelial lining cells were flattened. Their lumens were filled with eosinophilic material. Other alveoli were completely degenerated and atrophied. The homogeneous tissue was rather avascular except for occasional dilated venous sinusoids.

When stained with cresyl violet the major portions of the amyloid substance became a metachromatic pink. About the blood vessels the amyloid stained more deeply and appeared purplish. Between these two intensities of color were many gradations in metachromasia. The media of the arteries contained much amyloid. With congo red the major portion of the amyloid stained orange-red.

Course.—Several days later the Bennhold test was performed in an attempt to demonstrate the possible existence of a general amyloid involvement. Congo red, injected intravenously, showed 26 per cent retention of the dye in the tissue after one hour; this was considered to be within normal limits. On the day following injection of the dye laryngeal examination revealed the exuberances on the right laryngeal wall and on the anterior part of the left false vocal cord to be stained a deep pinkish red. This vital staining aided in the localization of the tumor from the surrounding tissue. On the following day a direct laryngoscopy was performed and the remaining amyloid tissue was removed. The voice promptly improved but was by no means normal. The laryngeal tissue appeared to be healing by healthy granulations. Because of the prominence of the lingual tonsil and lymphoid tissue at the base of the tongue a biopsy was made on material from this region. This failed to show any evidence of amyloid.

The patient was discharged much improved. There were no further collections of amyloid in the larynx grossly. She was referred to the radiotherapy department for further treatment to prevent recurrence.

COMMENT

The presence of an isolated amyloid tumor of the larynx without any demonstrable underlying local disease or general amyloid disease warrants the diagnosis of idiopathic amyloid tumor. The available clinical and laboratory observations in this case do not speak for the existence of a diffuse amyloid involvement. As is characteristic of local amyloid tumors of this type the deposits in the larynx were multiple, sessile and indefinitely demarcated from the surrounding structures. The mucosa was everywhere intact. That we were not dealing with a preexisting blastoma of the larynx is, however, evident from the microscopic picture, which revealed no evidences of a benign or malignant

new-growth. The amyloid mass extended from immediately beneath the epithelium to the deeper structure of the larynx and made up the entire tumor substance.

REVIEW OF THE LITERATURE

Tumor-like accumulations of amyloid in the upper air passages have been described by Schönhof, Herxheimer and Schmidt. The first comprehensive study of these amyloid tumor-like collections was made by Pollak in 1914.

TABLE 1.—*Cases of Local Idiopathic Amyloid Disease Reported in the Literature up to 1928*

Date	Author	Number of Cases	Total
1903	Herxheimer	24	24
1914	Pollak	26	50
1919	New	9	59
1925	Werner	7	66
1928	Schmidt	11*	77

* This does not include sixteen cases of secondary nodules.

TABLE 2.—*Cases of Local Idiopathic Amyloid Disease Reported in the Literature from 1928 to 1934*

Author	Date	Location
Noel and Aloin	1928	Nasal chamber
Petit and Moreau	1928	Base of tongue
Ecleo Huizinga	1928	Larynx
Juido Sotti	1928	Larynx
Wilhelm Hallermann (2 cases)	1928	Lung
Sauriez	1930	Tongue and larynx
von Soubiron	1930	Larynx
Bang and Blegvad	1932	Larynx
G. C. Müller	1932	Nasal chamber
Kriegsmann	1932	Larynx
Alfons Bauer	1932	Trachea
Krisper	1932	Tongue
Reuter	1932	Larynx
Gioacchini	1932	Larynx
Mutschler	1933	Tonsil
Beavis	1934	Larynx
Kramer and Som	1934	Larynx

The pertinent literature has since then been reviewed a number of times, and several additional cases have been reported. By confining ourselves to a strict definition of local idiopathic amyloid disease we have been obliged to exclude secondary amyloid deposits in laryngeal polypi and singer's nodes (Huebschmann; Peroni). The case of macroglossia reported by Gerstel is better classified with the group of atypical generalized amyloidosis. We have been able to collect seventy-seven true instances of idiopathic local amyloid tumors up to 1928 (table 1). From 1928 to the present we have been able to collect eighteen additional cases, including our own (table 2).

Localization.—The most frequent site of amyloid in the upper air passages is in the larynx. The next most common locations are the base

of the tongue, the trachea, the nasal cavity and the pharynx. The nasopharynx and lung are rarely involved. Most often the base of the tongue and larynx are involved simultaneously. Table 3 indicates the relative frequency of involvement of the upper air passages in the entire series of ninety-five cases.

Age and Sex.—Men were more frequently afflicted than women, the ratio being three to one. The usual age incidence was between 50 and 60 years, although occasional cases occurring in the second or third decade of life have recently been recorded (Kreigsmann, 21 years;

TABLE 3.—*Relative Frequency of Involvement of the Upper Air Passages*

Location	Number of Cases
Larynx	36
Tongue	16
Larynx and tongue	8
Trachea	13
Trachea and bronchus	4
Nose	6
Pharynx and buccal cavity	4
Lung	4
Nasopharynx	3
Tonsil	1
Total	95

TABLE 4.—*Age Incidence*

Age	Number of Cases
19 to 20 years	3
21 to 30 years	8
31 to 40 years	14
41 to 50 years	15
51 to 60 years	27
61 to 70 years	12
71 to 80 years	6
Not listed	10
Total	95

Müller, 20 years). The oldest patient was 74 years old (Peptit and Moreau). The patient in the present case, aged 19, is the youngest on record.

Symptoms.—Local amyloid tumors are often symptom-free, but sometimes they produce symptoms by mechanical pressure according to their size and location. Thus amyloid deposits at the base of the tongue are usually found accidentally at necropsy, whereas laryngeal lesions may produce hoarseness, stridor and even suffocation. Silent isolated deposits, unrecognized during life, were observed at postmortem examination by Krisper, Schmidt and Hallermann.

Onset.—Amyloid tumors have always been regarded as growing very slowly, generally requiring long periods of silent growth before manifesting themselves clinically. G. B. New stated that many years usually

elapse before the tumor attains sufficient size to permit recognition. However, that such deposition can take place in shorter periods has recently been proved by Mutschler who reported a case in which there was a definite history of only five weeks during which the amyloid accumulated in sufficient quantity to produce symptoms.

Hoarseness is by far the most common symptom in laryngeal lesions and is the result of improper approximation of the vocal cords owing to the presence of the tumor. Dyspnea and stridor are dependent on the relative size of the tumor and on its position in relation to the laryngeal aperture. The patients of Bauer and Kriegsmann required emergency tracheotomy for relief of impending suffocation. Johanni reported the occurrence of asphyxia and death in a patient as a result of torsion of a pedunculated amyloid tumor of the larynx.

Appearance.—The appearance of multiple waxy masses in the larynx is suggestive of local amyloid deposits, but the diagnosis is difficult without biopsy. The striking appearance of the lesion in the larynx in our case after the intravenous injection of congo red dye for detection of generalized amyloid disease suggested the application of the same test in the identification of localized forms of the disease clinically. Amyloid tumors in the larynx are usually multiple and show a tendency to involve the trachea and the base of the tongue. In most reported cases the tumor appeared waxy, dirty grayish or yellow, except when superimposed infection was present. The tumor was almost always covered with normal mucous membrane. Ulceration occurred only in the presence of severe infection, as in Bender's case in which intense pharyngitis coexisted, or following local trauma (Holmgren). Pain and adenopathy were present in the latter instance only as result of the associated infection.

Two distinct types of amyloid deposition in the upper air passages are recognized: (1) diffuse or submucous, and (2) nodular.

The submucosal type occurs more frequently in the trachea (Bauer) but occurs also in the larynx (Huizinga). In these sites it is prone to produce tracheal or laryngeal stenosis. The deposits are extensive and are never clearly demarcated from the surrounding structures. Complete removal of the tumor, even by thyrotomy, is extremely difficult.

The nodular type may be pedunculated or sessile. Instances of the former type in the larynx were described by New and Beavis. Such tumors may attain considerable size and give rise to distressing stridor because of the sudden dislodgment of the mass into the lumen of the larynx. The sessile variety is more common. This type of tumor is attached along a wide base and often blends with the surrounding structures. Frequently, smaller deposits are present at a distance from the main tumor in the larynx, and occasionally the lesion is bilateral (New; Kramer and Som).

Differential Diagnosis.—A diagnosis is usually impossible without biopsy, although intravital staining as done in our case may make the diagnosis highly probable. Cases of diffuse macroglossia caused by amyloidosis have been erroneously regarded as malignant, and the patients left untreated because the true nature of the tumor was not established.

Recurrence.—Pollak stated that recurrence is unavoidable even after total extirpation. Repeated recurrences were described by Evers, Berger, Werner and Giles. In Kriegsmann's case the tumor recurred after three attempts at complete removal. On the other hand, Huizinga and Pollak noted complete regression of amyloid after a simple removal of material for biopsy. An attempt was made to explain this on the basis of absorption of the amyloid following slight postoperative reaction.

Therapy.—Radical removal, preferably by direct or suspension laryngoscopy, offers the best chance for a permanent cure. Care should be exercised not to overlook small exuberances sometimes dispersed under the laryngeal mucosa. Vital staining with intravenous congo red delimited the borders of the tumor and was of definite aid to us in effecting total removal.

Many surgeons have had to resort to laryngofissure (Bauer; New; Gioacchini) for complete removal of the mass. When total extirpation is impossible even by thyrotomy, as in the case of extensive submucosal deposits, radiotherapy should be instituted. Mutschler used diathermy with good results. In 1922 Willimann obtained a permanent cure with radiotherapy. Similar cures were reported by New and Schmidt; their patients were free from recurrence after nine years. Evers, however, obtained no results after radiotherapy in two cases in which the deposits were not suitable for surgical removal. The value of radiotherapy in eradicating amyloid is still unsettled. Liver therapy has been tried; the results are still uncertain.

SUMMARY

Idiopathic or primary local amyloid tumors occur without corresponding amyloid deposits elsewhere in the body. They are to be distinguished from (1) amyloid deposits occurring secondarily in preexisting neoplasms or areas of chronic inflammation; (2) local tumor-like accumulations of amyloid occurring in the course of generalized amyloidosis, and (3) tumor-like amyloid masses in generalized amyloidosis with atypical localization.

The idiopathic amyloid deposits occur most frequently in the upper air passages, the larynx being the most favored site. These tumors are usually multiple and present a nodular, waxy appearance. Biopsy and intravital staining with congo red usually establish the diagnosis. Microscopically the picture is that of a homogeneous, structureless mass of

tissue that is readily identified as amyloid with the aniline stains. These tumor-like accumulations of amyloid may be without symptoms; when symptoms are present they depend chiefly on mechanical pressure. As recurrences are frequent, total extirpation is essential. When radical removal is not feasible, radiotherapy has been found to be of great value.

121 East Sixtieth Street.

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Clinical Notes

A LOW-COST PORTABLE AUDIOMETER

MATTHEW S. ERSNER, M.D.; EDWARD B. GLEASON, M.D.; GEORGE M. COATES, M.D., AND DAVID MYERS, M.D., PHILADELPHIA

We are presenting a new and inexpensive audiometer designed to replace the more costly ones which at present dominate the field of otology. We feel that this apparatus, because of its sturdiness, simplicity, dependability of operating, and last but not least, low cost, is an instrument worth bringing to the attention of otologists.

The development of the instrument was encouraged by Professor Gleason of Philadelphia. It is manufactured by the Brenco Electric Company of Philadelphia, and is designated by them as model 34B. It is made up in a leather carrying case and is small and compact, its total carrying weight being only 15 pounds (6.8 Kg.); it is always available, therefore, for ready transportation. It operates on a standard 110-120 volt A.C. current, but can be employed with a transformer on D.C. current as well. Thus, it is ready for operation on merely plugging it into the house socket, and one avoids the use of heavy "B" batteries, which are apt to be run down just when the apparatus is most needed.

In addition, this instrument has an oscillation range of from 64 to 8,192 cycles per second, divided into eight octaves of 64—128—256—512—1,024—4,096 and 8,192 (fig. 1). It has a potentiometer (fig. 1 *B-F*) to control the volume of sound passing into the receiver and is calibrated in sensation units. It has also a signal lamp (fig. 1 *G*) operated by a push button so that the patient may signal when the tone is audible or inaudible. Another feature is a tone interrupter (fig. 1 *E*) used to shut off all sound in the receiver. The Brenco audiometer has two tubes: one power and one oscillating tube.

It should be noted that, despite the technical details described, the low cost of the equipment brings it within the reach of every otologist.

During the past year we have been checking this instrument against other 2-A and 2-B audiometers on the market. A double record was made of each patient. It has been found that it checks accurately with other instruments within the limits of the equipment. Tests were made as follows:

(*a*) A series of one hundred medical students were tested, because the more intelligent the patient, the more accurate the test. In fact, it is common knowledge that with a certain class of patients it is impossible to utilize this instrument. Since the reaction time is an important factor in the test, we called on these subjects for their cooperation. It was found that the two instruments gave identical graphs for the one hundred students tested. Each one of the students presented practically a normal graph.

(*b*) We also tested patients from the otologic outpatient clinic of the Temple University Hospital and the Graduate Hospital of the University of Pennsylvania,

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From the Departments of Otology, Temple University School of Medicine, and the University of Pennsylvania Post-Graduate School of Medicine.

Fig. 1.—The parts of the new Brenco audiometer 34B are shown as follows: *A*, switch; *B*, potentiometer control; *C*, telephone plug-in; *D*, dial to vary intensity; *E*, tone interrupter; *F*, power indicator; *G*, signal button for patient; *H*, signal lamp; *I*, rubber sponge to fit over ear-piece for elimination of bone conduction; *K*, telephone head-piece, and *L*, dial for varying pitch.

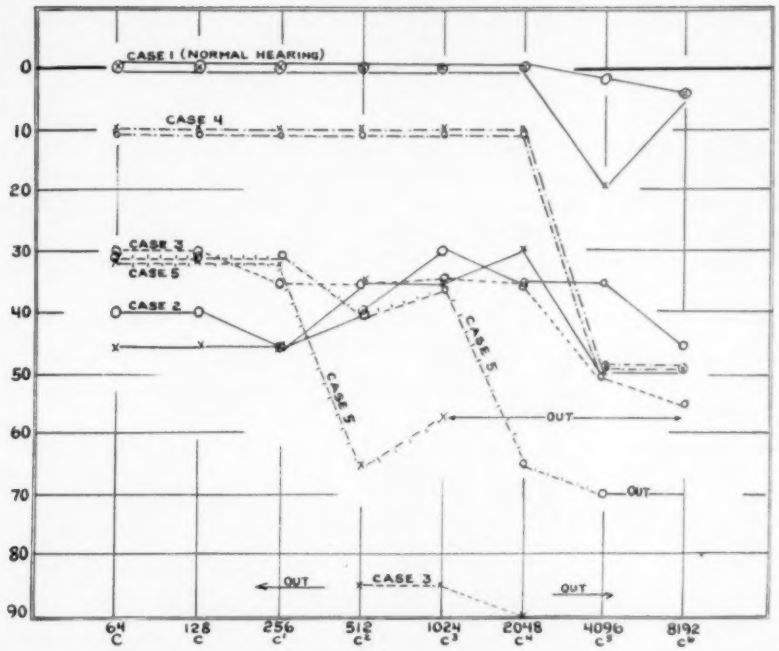

Fig. 2.—Curves of hearing obtained by the use of the Brenco 34B audiometer. In case 1, the subject had normal hearing; in case 2, there was a mixed type of deafness; in case 3, a mixed type of deafness in the right ear and practically a total loss of hearing in the left ear (abscess of the temporosphenoid lobe with recovery); in case 4, nerve deafness; in case 5, marked nerve deafness. In each case, the *o* curve represents the hearing in the right ear; the *x* curve, that in the left.

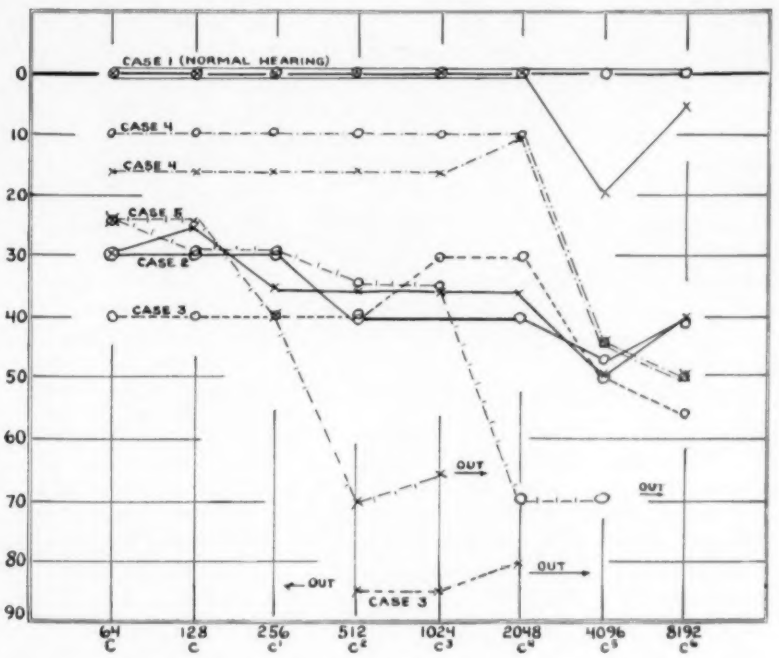

Fig. 3.—Curves of hearing obtained in the same cases as in figure 2, by the use of the standard audiometer 2-B.

as well as some from our practices. Composite charts are presented, giving five illustrative graphs; figure 2 illustrates curves obtained with the Brenco audiometer 34B, and figure 3, the comparison curves obtained by the use of the standard 2-B audiometer. As has been stated, these curves are practically identical, and therefore we believe that the new instrument may be depended on for accuracy.

SUMMARY

1. A new audiometer is described.

2. Cases are presented illustrating the accuracy of the instrument in comparison with other standard apparatus.

3. The low cost of the equipment and the ease of operation make it desirable and readily available.

1915 Spruce Street.

SUBLINGUAL EPITHELIAL CYST

Report of a Case

JOHN B. HITZ, M.D., MILWAUKEE

Sublingual epithelial cysts are of comparatively rare occurrence. Colp [1] in 1925 collected from the literature reports of thirty-two cases, not including those reported by Debonnelle [2] in 1908. Most of the cases had been reported in the European literature. Since 1925 I have been able to find only six cases reported in the American literature.[3]

I have used the term epithelial cysts purposely since Colp differentiated between a true dermoid cyst and an epithelial cyst which shows no skin appendages, such as hair, sweat glands and sebaceous glands.

Mikulicz stated that a dermoid cyst can develop in three ways: (1) through closure of the body cavities in the midline, (2) through closure of channels and clefts which were covered by epithelium during fetal life and (3) through abnormal deposits of epidermis in the deeper tissues. Colp stated that a dermoid cyst of the floor of the mouth probably can develop in all three ways.

Meyer [4] and Colp agreed that a true dermoid cyst is probably derived either from the closure of the mesobranchial field between the first and the second branchial arch or from abnormal remains of the tuberculum impar. Meyer was emphatic in stating that no epithelial cyst can be derived from the thyroglossal duct, as this is the true anlage of the thyroid gland and contains no epithelial ele-

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1. Colp, R.: Surg., Gynec. & Obst. **40**:183 (Feb.) 1925.

2. Debonnelle, E.: Les kystes demoïdes du plancher de la bouche, Thèse de Paris, 1908.

3. Cameron, J. R., and Boyko, G. V.: Dermoid Cyst of the Floor of the Mouth, J. A. M. A. **89**:1149 (Oct. 1) 1927. Meigs, J. V.: New England J. Med. **198**:282 (March 29) 1928. Figi, F. A., and Harrington, S. W.: S. Clin. North America **9**:89 (Feb.) 1929. Joseph, M.: Am. J. Surg. **16**:509 (June) 1932. Meyer, H. W.: Ann. Surg. **95**:1 (Jan.); 226 (Feb.) 1932.

4. Meyer, H. W.: Ann. Surg. **95**:1 (Jan.); 226 (Feb.) 1932.

ments; furthermore, never at any time in the development of the human embryo is it a true duct. The lingual duct, according to Meyer, develops from the drawing of epithelial elements into the deeper tissues as a result of the descent of the anlage of the thyroid gland. This duct often gives rise to small cysts of the body of the tongue and could be the origin of a sublingual cyst. A lingual cyst usually contains either stratified squamous or ciliated epithelium or both. Colp, in his

Photomicrograph of a section through the sublingual epithelial cyst.

report of thirty-two cases, stated that nine cases were not accompanied by a pathologic report of the wall of the cyst. In five cases the cyst showed none of the characteristic appendanges of the skin except stratified squamous epithelium; these cases, he believed, were instances of a cyst derived from the lingual duct.

Further details of the embryologic derivation, the diagnosis and the treatment, are given in the two excellent articles by Colp[1] and by Meyer.[4] Suffice it to say here that sublingual cyst is believed always to be congenital, though it may

not become apparent until between the eighteenth and the twenty-fifth year. It is usually situated in the midline, but it may be lateral. It is usually not accompanied by symptoms until it is sufficiently large to interfere with speech and with swallowing. Treatment consists of complete excision of the cyst either through the mouth or by incision in the submental region. General anesthesia must be used with caution in order to avoid suffocation as a result of having the tongue pushed back by the tumor.

REPORT OF CASE

E. D., a woman, aged 19, came to my office on June 25, 1933, complaining of a swelling under the tongue which was beginning to restrict the movements of the tongue and thereby to affect her speech. The growth had been present for one year. Examination revealed a large, smooth, fluctuant swelling in the midline and under the tongue. It was apparently submucous and was thought at first to be a ranula. However, Wharton's ducts were found to be patulous and functioning normally. When the patient closed her mouth a large, round swelling appeared in the submental region.

An operation was performed under local anesthesia on June 27. The lingual nerves were blocked at the angles of the jaw, and a transverse incision was made anterior to Wharton's ducts. A few atrophied muscle fibers were encountered. As the dissection progressed the lingual vessels and nerves were exposed, and procaine hydrochloride was injected into the latter. The cyst, which was found to be attached to the body of the hyoid bone, was removed *in toto*. The wound was closed with silk and with one deep catgut suture, and a rubber tube drain was left in place. Recovery was uneventful. The drain was removed in three days, and the sutures, in four.

The cyst measured 6 by 4.5 by 4 cm. Its walls were thick and enclosed a soft, putty-like mass. It appeared grossly to be either a dermoid or a sebaceous cyst.

Microscopic examination showed that the wall was lined by stratified squamous epithelium with marked hornification. Perivascular infiltration of lymphocytes was noted. In the center of the wall of the cyst was a hornified half-necrotic area. No evidence of hair follicles or of sweat or sebaceous glands was found in the sections examined. The contents of the cyst consisted of desquamated epithelial cells and a good deal of fatty sebaceous material, but no hair.

COMMENT

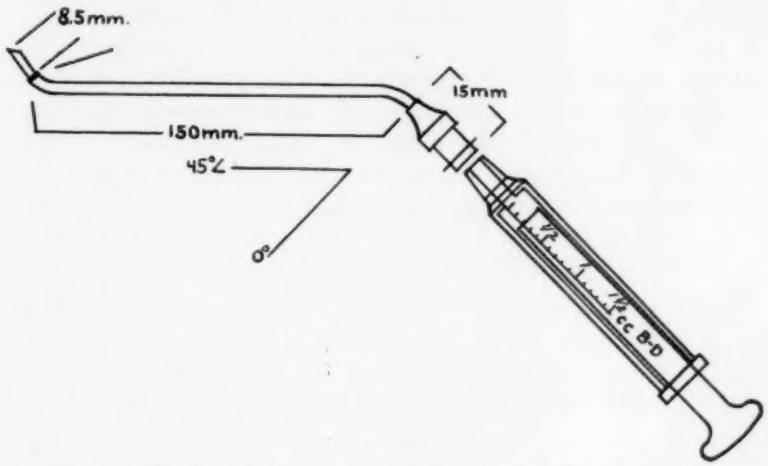
The derivation of a true dermoid cyst of the floor of the mouth seems to be well established, but whether an epithelial cyst without true skin appendages belongs in this group of true dermoids or is formed from the lingual duct still remains an open question which can be answered only by thorough histologic study in numerous cases. Reports of cases should always be accompanied by photomicrographs, and whenever possible the tumors should be studied in serial section. Unfortunately, in the present case the specimen was destroyed before complete serial sections were made. However, a sufficient number of slides were studied to warrant the conclusion that the cyst belonged in the same class as those in Colp's five cases, which he believed had their origin in the lingual duct.

208 East Wisconsin Avenue.

NEW NEEDLE FOR INJECTION INTO THE NASOSPHENOPALATINE GANGLION

GAIL E. CHANDLER, M.D., MIAMI, FLA.

I designed this instrument with three distinct advantages in view: 1. The measured 8.5 mm. needle is to assure safety against injury to the sphenopalatine ganglion. 2. The safety shank permits insertion only of the measured needle. 3. On connecting a loaded 1.5 cc. Yale syringe to the sphenopalatine needle before use, slipping of the needle is prevented by the 45 degree angle of the proximal end, which allows continuous free vision of the nasal area and makes it possible to hold the instrument in the proper place during the injection.



New needle for injection into the nasosphenopalatine ganglion.

TECHNIC

The usual method of sterilization is observed. Enlarged turbinates are reduced by spraying if necessary. The area over the sphenopalatine fossa is first thoroughly anesthetized by means of a cotton-wrapped applicator moistened with a suitable local anesthetic such as 10 per cent solution of cocaine or 1 per cent solution of p-n-butylaminobenzoyl-dimethylamino-ethanol hydrochloride. The needle, with the attached 1.5 cc. syringe containing 1 cc. of 1 per cent solution of procaine hydrochloride is introduced into the sphenopalatine fossa and held firmly while the attending nurse injects the drug. The needle is then removed, and from seven to ten minutes are allowed for a good anesthesia to take place. The injection of procaine hydrochloride may be omitted if desired. Then the procedure is repeated, 1 cc. of alcohol being used. Administration of two tablets of acetylsalicylic acid adds to the patient's comfort.

This instrument is made and stocked by Becton, Dickinson and Co., Rutherford, N. J.

501-2 Huntington Building.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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PERORAL ENDOSCOPY

LOUIS H. CLERF, M.D.
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In no field of medicine has greater progress been made in recent years than in that pertaining to the diagnosis of neoplasms involving the tracheobronchial tree. Bronchoscopy can lay claim to much of the credit. In the era prior to the use of the bronchoscope in the diagnosis of bronchopulmonary conditions knowledge of tumors of these structures was based largely on observations at autopsy.

NEOPLASMS OF THE TRACHEA AND BRONCHI

Tumors of the Trachea.—Primary tumors of the trachea are uncommon, particularly in children. Richards and Dietrich [1] reported the occurrence of primary fibrosarcoma of the trachea in an 8 month old infant. The symptoms consisted of wheezing and dyspnea of one week's duration. Tracheotomy was performed, with considerable relief of the dyspnea. The first bronchoscopic examination showed no abnormality; however, at a subsequent examination narrowing of the left bronchus was found. Death occurred rather suddenly about six months later, and a definite diagnosis was made at autopsy. A mass was observed incorporated in the tracheal wall, extending to the left main bronchus, with involvement of the lymph nodes at the bifurcation of the trachea. It was the opinion of the pathologist that the tumor was primary in the tracheal wall.

In their record of a case of primary adenocarcinoma of the trachea, Simpson and Moore [2] emphasized the importance of bronchoscopy as a diagnostic aid. They found that but twenty-seven cases of primary carcinoma of the lower third of the trachea had been reported in the literature. In a majority of these the diagnosis was made

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1. Richards, L. G., and Dietrich, H. F.: Fibrosarcoma of the Trachea, Ann. Otol., Rhin. & Laryng. **43**:892 (Sept.) 1934.

2. Simpson, W. L., and Moore, R. M.: Primary Colloid Adenocarcinoma of the Lower Third of the Trachea, Ann. Otol., Rhin. & Laryng. **43**:1133 (Dec.) 1934.

late in the disease. They are agreed that the occurrence of dyspnea in a person apparently in good health and in the absence of signs of intrathoracic disease are suggestive of tracheal tumor and warrant diagnostic bronchoscopy.

Inflammatory Tumors of the Bronchi.—In a study carried out to ascertain the etiology of inflammatory tumors of the bronchi, Peroni [3] examined the records of patients for a possible cause of the formation of these growths, attempted to reproduce these tumors experimentally by producing in animals the conditions which may be presumed to produce or favor the genesis of such lesions and tried to discover the etiologic explanation from a histologic study of the growths by comparing them with inflammatory or degenerative hyperplastic conditions in other organs. He succeeded in producing only the type of inflammatory tissue changes that are commonly found in pulmonary suppuration and in the presence of foreign bodies in a bronchus. On the basis of comparative histologic studies of tumors and inflammatory tissues, he expressed the opinion that these tumors are in most cases not produced by the action of pus derived from suppurative bronchopulmonary processes. He believed that the suppurative lesions often observed in association with these tumors of the bronchi are more rationally to be regarded as secondary to the obstruction. Bronchial lesions with tumor formation seem to consist of subepithelial hyperplastic tissue which may represent a herniation of chronic inflammatory tissue into the bronchial lumen caused by processes of carnification of the lung.

Primary Carcinoma of the Bronchus.—Rabin and Neuhof [4] shared the opinion held regarding the value of bronchoscopy in determining the location and the type of neoplasm. In their opinion, visualization of a neoplasm through a bronchoscope is usually indicative of a noncircumscribed infiltrating growth, and nonvisualization is indicative of a circumscribed neoplasm. In their experience, the great majority of localized neoplasms of a branch bronchus have been situated too far down the bronchial tree to be seen. The noncircumscribed type, to which practically three fourths of pulmonary cancers belong, comprises tumors growing from main and from branch bronchi. These neoplasms are termed tumors of the main bronchus and tumors of the branch bronchus. They present the usual invasive characteristics of cancer in the great majority of cases. The great

3. Peroni, A.: Inflammatory Tumors of the Bronchi: Experimental and Pathologic Considerations, Arch. Otolaryng. **19**:1 (Jan.) 1934.

4. Rabin, C. D., and Neuhof, H.: A Topographic Classification of Primary Cancer of the Lung: Its Application to the Operative Indication and Treatment, J. Thoracic Surg. **4**:147 (Dec.) 1934.

proportion of tumors of the main bronchus and tumors of the branch bronchus, with the exception of the peripheral type, do not fall into the operable class, in the sense of surgical eradication, at the time at which patients suffering from the lesion ordinarily come under observation.

Arbuckle [5] discussed several classifications of pulmonary cancer with regard to their anatomic and histologic application and briefly emphasized the importance of securing a comprehensive history, particularly with regard to the development of symptoms. He also directed attention to the peculiar type of cough that is observed in certain of these cases. He agreed that it is now generally conceded that after all other methods of study have been carried out the final evidence on which a diagnosis must rest, if it is to be made early enough in the course of the disease to permit of successful treatment, must be produced through the efforts of the bronchoscopist, namely, biopsy of tissue removed bronchoscopically. This procedure is not only of value as a diagnostic aid, but is often a means of securing additional information that may be of importance in treatment.

In their presentation, based on a review of thirty-two cases of bronchopulmonary cancer in which the bronchoscopic findings were confirmative, Jackson and Konzelmann [6] concluded that bronchoscopic biopsy gives positive results in about 75 per cent of the cases of bronchial carcinoma. In many cases in which biopsy is not possible at the first bronchoscopy the patients later show endobronchial intrusion, though the surgeon should not wait for this if consent is given for exploratory thoracotomy. Even in cases in which biopsy is not possible, bronchoscopic findings may be of definite diagnostic value. A simple classification of bronchiogenic carcinoma that can be understood and followed by the pathologist, bronchoscopist and radiologist is desirable. Microscopic evidence of malignancy does not imply necessarily an extensive infiltrative tumor, and therefore the grading of bronchiogenic carcinoma should be tempered by a knowledge of the bronchoscopic and roentgenoscopic findings. Biopsy studies are of great value, yet constitute but one of the important considerations in the diagnosis of bronchiogenic tumors. Team-work by the bronchoscopist, the radiologist and the pathologist will lead to a proper prognosis and guide the patient to the most beneficial form of treatment.

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5. Arbuckle, M. F.: Clinical Diagnosis of Primary Cancer of Lung, Ann. Otol., Rhin. & Laryng. **43**:1174 (Dec.) 1934.

6. Jackson, C. L., and Konzelmann, F. W.: Bronchial Carcinoma: Bronchoscopic Biopsy in a Series of Thirty-Two Cases, J. Thoracic Surg. **4**:165 (Dec.) 1934.

In a discussion of cases of pulmonary disease in which bronchoscopy had been employed with usefulness from the standpoint of both diagnosis and treatment, Zamora [7] cited his experiences with twenty consecutive patients who were sent for bronchoscopic examination and in whose cases a presumptive diagnosis of bronchial carcinoma had been made. In twelve of these, specimens secured bronchoscopically were positive on microscopic examination. Since histologic examination is the only conclusive test and as the results are positive in so large a percentage of cases, bronchoscopic examination of every patient with a suspected case is a logical diagnostic procedure.

Melanoma of the Bronchus.—In reporting a case of melanoma of the bronchus, Clerf [8] emphasized the importance of recognizing wheezing respiration as a sign of partial obstruction of a bronchus and the important part that bronchoscopy must necessarily play in the diagnosis of obscure symptoms and signs in the chest. In the case reported there was obstructive atelectasis involving an entire lung. At bronchoscopy an obstructing growth was removed and the atelectasis relieved. After receipt of the histologic report that the lesion was a melanoma, a history of an almost forgotten removal of a pigmented mole from the arm was obtained. Repeated bronchoscopic removal of the growth aided materially in prolonging life, although the prognosis was hopeless.

In discussing the problems of diagnosis of carcinoma of the lung, Peroni [9] agreed with the views generally expressed regarding the importance of bronchoscopic examination in the early diagnosis of carcinoma. He emphasized the importance of making the diagnosis early in the disease, for only in this way can surgical treatment be considered. Attention was directed to the occurrence of suppurative processes of the lung secondary to neoplastic obstruction to a bronchus, and the importance of diagnostic bronchoscopy in these cases in order to establish a definite diagnosis was stressed.

In a study of the physics and mechanics of the intrathoracic viscera, especially the lungs, comparison with the ordinary bellows may be made. In discussing the factors in check-valve obstruction, the Jacksons [10] directed attention to the bronchoscopically demonstrated

7. Zamora, A. M.: Notes on Bronchoscopy with Examples of Its Application, Lancet **2**:864 (Oct. 20) 1934.

8. Clerf, L. H. : Melanoma of Bronchus: Metastasis Simulating Bronchogenic Neoplasm, Ann. Otol., Rhin. & Laryng. **43**:887 (Sept.) 1934.

9. Peroni, A.: The Bronchoscopic Diagnosis of Cancer of the Lung, Atti e mem. Soc. lomb. di med. **1**:10, 1934.

10. Jackson, C., and Jackson, C. L.: Bronchial Obstruction, with Special Reference to Endobronchial Tumors, Pennsylvania M. J. **37**:740 (June) 1934.

fact that a tumor may, by valvular action, cause obstructive emphysema or obstructive atelectasis. While this is generally accepted, there are other factors entering into the mechanism that are not so well understood. One or more of the following mechanisms may be concerned: Inspiratory increase and expiratory decrease of the lumen are fundamental to all forms of check-valve action. Narrowings of the bronchial lumen are relative. A pedunculated tumor may flop to and fro in the current of air, causing emphysema or atelectasis according to "the set of the valve." A sessile or annular tumor may diminish the lumen to such a degree that it is obliterated early during the expiratory phase, producing emphysema. Peribronchial lesions may compress the lumen to the point of setting up valvular action. This may occur as an adenopathic compression or involvement of the bronchial wall sufficient to produce any of the three types of bronchial obstruction. Inflammatory changes in the bronchial wall, granulations, normal or pathologic, secretions, blood clots, crusts and more or less solid exudates may either assist or be the sole mechanism.

Stevens and Hudson [11] presented a group of cases illustrative of the different types of bronchial obstruction. They were agreed that bronchial obstruction can be dealt with as a definite entity. The causative influences are varied and many lie within the bronchial lumen or within the wall of the bronchus or may be entirely extrabronchial. The treatment must be varied in accordance with the causes. Early diagnosis and treatment are highly essential, and good results are dependent on one's ability to relieve or remove the etiologic factors.

BRONCHOSCOPY IN TUBERCULOSIS

Pulmonary tuberculosis, long considered by many as a "no man's land" for bronchoscopists, is now being studied by direct methods in the presence of certain well defined indications. As a result certain obscure conditions have been clarified, and a comprehensive literature of the bronchoscopic aspects of the problem is being accumulated.

In an excellent study of the development of bronchial stenosis in pulmonary tuberculosis, Eloesser [12] summarized his findings as follows: Stenosis of the air passages from the trachea to the bronchioles is not infrequently due to various tuberculous processes. These processes, their anatomy and their affects are described. The clinical picture of stenosis at various levels and sites is discussed. The clinical, roentgenographic and bronchoscopic signs are presented, and an attempt is made to explain

11. Stevens, R. H., and Hudson, W. A.: Bronchial Obstruction: Its Diagnosis and Treatment, Radiology **22**:339 (March) 1934.

12. Eloesser, L.: Bronchial Stenosis in Pulmonary Tuberculosis, Am. Rev. Tuberc. **30**:123 (Aug.) 1934.

them. The treatment of stenosis is briefly discussed. Eloesser expressed the hope that the attention of those interested in tuberculosis might be drawn to this permanent, therapeutically important but rarely diagnosed lesion.

Myerson [13] expressed the opinion that certain general indications for bronchoscopy in the nontuberculous person may be applied to the tuberculous person. In addition, there are certain indications which may be considered as directly related to the disease. Under this group may be considered those cases of pulmonary tuberculosis presenting obstructive phenomena, hemoptysis of unexplained origin and asthmatic breathing. In addition, bronchoscopy may be indicated for localization of the disease and the securing of additional data for the surgeon and, under certain conditions, for the securing of a positive bacteriologic specimen from the branch bronchi which bring exudate into the main bronchus. Myerson analyzed a series of sixty patients who were either known to have pulmonary tuberculosis or were strongly suspected of having this disease. These were subjected to bronchoscopy because of a definite indication in the minds of the referring clinicians, all of whom were experts in the field of diseases of the chest. The conditions which were encountered are described; a number of cases are reported, and the bronchoscopic findings are cited. This investigation strongly indicates that bronchoscopy should have a definite place in the study of the tuberculous patient. No untoward results were observed by Myerson as a result of bronchoscopy in pulmonary tuberculosis.

Having demonstrated experimentally that bronchial occlusion could be produced by cauterization of a bronchus with a 35 per cent solution of silver nitrate and that the atelectasis of pulmonary tissue brought about by bronchial occlusion exerted a favorable influence on experimental pulmonary tuberculosis in dogs, Adams and Vorwald [14] decided to test its application in cases of tuberculosis in man. In four hopeless cases with bilateral involvement the cauterizing agent was applied a total of twelve times. Obviously there was little hope of benefit because of the extent of the disease. The work seemed to demonstrate conclusively that the bronchi could be closed in man as readily as in dogs; also, that little or no reaction attended the application of the cauterizing agent in man.

In experimental studies on endobronchial occlusion, Cutler and Wood [15] failed to secure satisfactory results following the use of silver

13. Myerson, M. C.: Bronchoscopy in Tuberculosis, Ann. Otol., Rhin. & Laryng. **43**:1139 (Dec.) 1934.

14. Adams, E. W., and Vorwald, A. J.: The Treatment of Pulmonary Tuberculosis by Bronchial Occlusion: An Experimental Study, J. Thoracic Surg. **3**: 633 (Aug.) 1934.

15. Cutler, E. C., and Wood, C. B.: Studies on Endobronchial Occlusion, Surg., Gynec. & Obst. **59**:501 (Sept.) 1934.

nitrate solutions, a solution of glacial alcohol in absolute alcohol and electrical cauterization as stenosing agents. Experimental studies with acriflavine seemed to indicate that this would be a safe and satisfactory agent. A 25 per cent aqueous solution of acid acriflavine as used in these experiments produced aseptic necrosis of the bronchial mucosa and submucosa and the formation of a firmly adherent mucoid plug. Later the rapidly regenerating epithelium separated the plug from the walls, and this was coughed out. The peribronchial and submucosal tissues proliferated, became fibrosed and occluded the bronchial lumen. Pulmonary atelectasis occurred only when all the bronchi to the lobe had been completely occluded. Permanent pulmonary atelectasis, even of a whole lung, was compatible with the normal health and good general condition of the experimental animal.

BRONCHOSCOPY IN OTHER DISEASES

Elimination of the lingual death zone is fundamental, according to the Jacksons,[16] to avert impending death by asphyxia. This zone anatomically includes the funnel-like pharynx and the tongue, which on dropping back fills that funnel, acting as a check-valve. The best way to eliminate the check-valve is to lift the tongue forward from the wall of the pharynx, using direct laryngoscopy. A bronchoscope may then be introduced through the laryngoscope into the larynx and trachea. Aspiration of secretion from the patient's pharynx and tracheobronchial tree through the laryngoscope and bronchoscope prevents him from drowning in his own secretions. The equipment necessary for the prevention of asphyxial death should be available in every ambulance and every emergency ward. This consists of one direct laryngoscope, one bronchoscope with a full lumen aspirating pattern of 5 mm. by 30 cm., one tank of oxygen containing a 5 per cent mixture of carbon dioxide and one piece of rubber tubing about 4 feet (1.2 meter) long attached to the outlet of the tank.

Diphtheria.—In dealing with obstruction of the larynx, trachea and bronchi occurring in diphtheria, Bailey[17] recommended using an anterior commissure laryngoscope for exposure of the larynx. Pseudomembrane should be removed, using an aspirating tube. Removal of this does not leave a bleeding mucosa. The aspirating tube is then inserted gently down through the larynx into the trachea and also into the bronchi, if necessary, and aspiration continued until no membrane is obtained. A bronchoscope should not be passed in a case of this kind; a blunt aspirat-

16. Jackson, C., and Jackson, C. L.: The Lingual Death Zone in Aphyxia, New York State J. Med. **34**:15 (Aug. 1) 1934.

17. Bailey, C. W.: Modern Surgery in Diphtheria: Observations on Six Thousand and Eleven Cases, Arch. Otolaryng. **20**:162 (Aug.) 1934.

ing tube from 20 to 40 cm. long used through an anterior commissure laryngoscope is much better, because the bronchoscope is of much larger caliber than the aspirating tube, and consequently there is more danger of traumatizing the inflamed laryngeal mucosa. The usual postbronchoscopic laryngeal edema would be a serious consequence in these cases, whereas there is no such reaction from the use of the aspirating tube through the laryngoscope. The pseudomembrane can be removed from the larynx, trachea and large bronchi just as well and possibly better with the blunt aspirating tube alone than when it is used through a bronchoscope. The use of the aspirating tube makes possible much quicker removal and therefore greatly lessens the fatigue to the patient. This is an important consideration.

Syphilis.—A case of tracheoesophageal fistula of syphilitic origin was reported by Lukens and Ono.[18] No difficulty was encountered in visualizing the opening of the fistula on the anterior wall of the esophagus by esophagoscopy. The opening was about 2 cm. in diameter, but was practically completely obstructed by a necrotic flap. A new clinical sign, the Ono sign, which may be elicited in cases of fistula between the esophagus and airway, is described.

Direct evidence of syphilis of the lung secured bronchoscopically is rarely obtained ante mortem. In the case reported by Hart and Gibbon,[19] a tentative diagnosis of syphilis was made before the serologic findings were reported. This was based on histologic examination of tissue removed bronchoscopically from the left bronchus. In this locality an indurated mass was found which appeared to occlude the bronchus leading to the lower lobe of the left lung. The subsequent course following active antisyphilitic treatment, with definite evidences of clearing of the pulmonary lesion, seemed to corroborate the diagnosis of syphilis. The facts in this case emphasize the importance of bronchoscopy in obscure lesions of the chest.

Status Lymphaticus.—Certain bronchoscopic observations on status lymphaticus in the adult were recorded by Soulas.[20] Studies were carried out in the course of routine bronchoscopic treatment of bronchial and pulmonary suppurations, particularly in cases of tracheobronchitis, bronchorrhea with bronchial dilatations and a few cases of asthma. Generally speaking, the appearance of the tracheobronchial tree in the lymphatic subject was characteristic. There was a tendency in these

18. Lukens, R. M., and Ono, J.: Syphilitic Tracheoesophageal Fistula; Report of Case, Laryngoscope **44**:334 (April) 1934.

19. Hart, V. K., and Gibbon, J. W.: Syphilis of the Lung, South. Med. & Surg. **96**:404, 1934.

20. Soulas, A.: The Point of View of the Bronchoscopist Regarding Status Lymphaticus in the Adult, Bronchoscop., oesophagoscop. et gastroscop. **3**:253 (July) 1934.

cases to the production of a great deal of pus, also to stagnation of secretions. The mucosa was usually pale and somewhat thickened. The reaction to epinephrine was slight. From the standpoint of the respiratory mechanism, it was observed that bronchial movements were limited. The cough reflex was delayed or slow. Persons with this condition are throat clearers rather than coughers. From a technical point of view, these subjects were easier to examine, as the bronchial tubes were widely dilated and presented sluggish motor reactions. The difference between expiratory contraction and inspiratory dilatation was very slight. Bronchial dilatations usually were of great dimensions, and secretions accumulated easily. In persons with asthma one found precisely the opposite of what was just observed; that is, there were violent spasmodic reactions with severe cough, rendering endoscopic maneuvers difficult. The violent movements of the trachea and bronchi simulated a real paroxysm. The mucosa was almost always hyperemic and bled easily, and the bronchial orifices were the seat of inflammatory stenosis. Secretions generally drained poorly and were usually less abundant than in status lymphaticus, but more tenacious and adherent. One must be careful in giving cocaine to these patients, as there seemed to be a greater risk of syncopal accidents in the cases reported. Weak anesthetics gave sufficient relaxation. Certain substances introduced directly into the bronchi had a beneficial influence. Iodized poppy-seed oil 40 per cent gave excellent results. In many of the patients it was superior to bronchial aspiration. Medication with arsenic and sulphur was found very efficacious.

Tucker [21] reported a number of cases of pulmonary disease in which bronchoscopy played an important rôle in arriving at a correct diagnosis or in carrying out appropriate treatment. The bronchoscopic aspects are reported in detail. He concluded that, owing to the perfection of instrumentarium and of the method of bronchoscopic procedure without general anesthesia, bronchoscopy had reached a point in development where it was invaluable as an aid in the diagnosis and treatment of pulmonary disease. Bronchoscopy should supplement all other methods of examination in pulmonary disease. In order to obtain the best results, special training and continuous practice by the bronchoscopist are required, together with a trained personnel, an adequate instrumentarium and the support and close cooperation of the internist, the roentgenologist and the surgeon. Modern bronchoscopy has created world-wide interest because of its efficiency as an aid in the diagnosis and treatment of pulmonary disease. Its continued advance and development will depend on the interest and support given by the internist and the pediatrician,

21. Tucker, G.: Bronchoscopy in Pulmonary Disease; Present Status an Aid in Diagnosis and Treatment, Ann. Int. Med. **8**:444 (Oct.) 1934.

who see the patients first and who decide whether or not they shall have the benefit of bronchoscopic consultation.

In a discussion of the treatment of pulmonary abscess based on a series of twenty-five consecutive cases, Cummings [22] agreed that the best results are secured by the cooperation of the internist, the bronchoscopist, the surgeon, the bacteriologist and the roentgenologist. Bronchoscopic aspiration is the most valuable form of therapy, second to postural drainage. It is also of value in differential diagnosis and in conjunction with roentgenography in the localization of the abscess.

In considering the treatment of pulmonary suppuration in which are included pulmonary abscess, as well as other types of pathologic changes of the lung in which necrosis is not grossly evident, Kearney [23] concluded that bronchoscopic aspiration of secretion is useful to prevent asphyxiation. He also expressed the belief that it is of value in preventing the spread of pneumonitis in nontuberculous suppuration. Diagnostic bronchoscopy should be performed in every case of bronchial obstruction, also in cases of nontuberculous pulmonary abscess, to rule out endobronchial neoplasms or the presence of a nonopaque foreign body.

Bronchoscopy was considered by Marvin [24] as a very important aid in the diagnosis of bronchitis. It is much more important and necessary from a therapeutic standpoint, as it is the means by which hemorrhage may be controlled or purulent secretion aspirated in patients in whom drainage by postural methods is insufficient. The greatest benefit from bronchoscopic treatment was secured in patients with secondary pneumonitis about the areas of bronchiectasis. In these, bronchoscopic aspiration may be the only effective method to prevent the spread of the inflammatory reaction, with possible abscess formation and death.

Advances in the investigation of the bronchial tree and in roengenography have, according to Lord,[25] contributed much toward the diagnosis and treatment of bronchiectasis. Recognition of this disease in the early stages makes it apparent that it is more common as an independent disorder than was formerly appreciated. In Lord's opinion, bronchoscopy is indispensable for the determination of bronchial obstruction. It may also be helpful in determining the source of blood or pus. In this author's experience, instillation of iodized oil into the trachea after evacuation by postural drainage gave results which were as satisfactory as those secured by introduction through the bronchoscope.

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22. Cummings, G. O.: Treatment and Management of Nontuberculous Pulmonary Abscess, Arch. Otolaryng. **19**:684 (June) 1934.

23. Kearney, H.: Bronchoscopic Aspiration in the Treatment of Lung Suppuration, New Orleans M. & S. J. **87**:169 (Sept.) 1934.

24. Marvin, H. P.: The Importance of Bronchoscopy in Bronchiectasis, Ann. Int. Med. **7**:903 (Jan.) 1934.

25. Lord, F. T.: Certain Aspects of Bronchiectasis with Special Reference to Diagnosis and Treatment, New England J. Med. **211**:437 (Sept. 6) 1934.

In considering bronchiectasis from the standpoint of treatment, Goodman [26] concurred in the views generally expressed by bronchoscopists that bronchoscopic treatment must be considered important in the removal of local causes, such as foreign bodies, in dilating strictures and in aspirating material from dilated bronchi to promote drainage. It seemed doubtful whether bronchoscopic treatment alone could cure bronchiectasis. It does, however, give temporary improvement in a certain number of cases.

DISEASES OF THE ESOPHAGUS

Congenital Lesions.—In a review of the literature, Patterson [27] found that congenital cyst of the esophagus is rare. She reported a case of cyst found post mortem in a child aged 7 months. When first observed, the child exhibited a typical clinical picture of malnutrition. There was an inspiratory crow. The findings by direct laryngoscopy were those commonly observed in cases of malnutrition, namely, a flabby, undeveloped larynx. Three days later the patient had dyspnea, with cyanosis and vomiting. A diagnosis of bronchopneumonia was made; death resulted within three days. At autopsy a cyst was found firmly attached to the esophagus, partly between the trachea and esophagus, but not communicating with either. Patterson emphasized the question of diagnosis. Unless there are symptoms of pressure or obstruction, diagnosis is difficult.

In a discussion of diaphragmatic hernia, particularly with reference to the type in which the esophagus is congenitally shortened, Dr. Haroen and Dr. Gerlings [28] reported the case of a girl, aged 3 years, who had symptoms referable to the esophagus, which had begun one week after birth. The roentgen findings were not conclusive. At esophagoscopy there was found stenosis of the esophagus at a point proximal to the hiatal level, together with an area of ulceration covered with flat granulations. No tissue was removed for biopsy, but these authors believed that the ulcer was of peptic origin. It healed promptly following topical applications through the esophagoscope.

Additional cases of congenital shortening of the esophagus were reported by Clerf and Manges.[29] Of fourteen patients with congenital

26. Goodman, H. I.: Suppurative Bronchiectasis; Its Surgical Treatment, Am. J. Surg. **26**:543 (Dec.) 1934.

27. Patterson, E. J.: Congenital Cyst of the Esophagus: Report of Case, Ann. Otol., Rhin. & Laryng. **43**:884 (Sept.) 1934.

28. Haroen, I. L., and Gerlings, P. R.: Congenital Short Esophagus with Peptic Ulcer of the Esophagus, Acta oto-laryng. **19**:461, 1934.

29. Clerf, L. H., and Manges, W. F.: The Congenitally Short Esophagus, J. A. M. A. **102**:2008 (June 16) 1934.

shortening of the esophagus with stenosis and the presence of a portion of stomach in the thoracic cavity, four were children and ten adults. Two children were brother and sister. Two groups of symptoms were noted, one consisting of those resulting from esophageal obstruction and malnutrition; in the other, symptoms due to ulceration were prominent, although there was some mechanical obstruction. The roentgenographic and esophagoscopic changes were those of narrowing of the lumen at the esophagogastric junction, a point above the normal hiatal level joined with a portion of stomach above the diaphragm. In several patients superficial ulceration of the mucosa was observed at the level of the stenosis. In eight cases mucosa was removed from the food passage above the level of the diaphragm for histologic examination. This proved to be gastric mucosa.

The hopeless outlook for present methods of treatment of the common form of atresia of the esophagus suggested to Iglauer [30] that a two-stage operation might be feasible for its relief. This consists of, first, cervical esophagostomy for drainage of a blind esophageal pouch and, at the second stage, a transthoracic operation for the externalization of the lower part of the esophagus, i. e., dorsal esophagostomy for purposes of feeding.

Spasm of the Esophagus.—Moersch and Camp [31] recorded their observations on a small group of patients who complained of intermittent dysphagia and who exhibited roentgen evidence of diffuse spasm of the lower third of the esophagus. The outstanding feature, from a roentgenographic standpoint, is the small degree of dilatation occurring above the affected portion, regardless of the duration of symptoms. This serves to distinguish it from typical cardiospasm. The esophagoscopic findings indicated that the lower end of the esophagus was spastic and that the normal movements of the esophageal wall during respiration were absent. In one patient a small area of superficial ulceration was noted immediately above the cardia. Application of silver nitrate in this area produced pain which was referred to the right shoulder. Pain was frequently pronounced and was commonly referred to the lower half of the sternum, extending through into the back. The exact etiology of the spasm is not known. No plan of adequate treatment has been decided on.

Mosher,[32] in discussing fibrosis of the esophagus and two cases of stricture of the esophagus, described four instruments employed in

30. Iglauer, S.: A Proposed Operation for the Relief of Congenital Atresia of the Esophagus, Ann. Otol., Rhin. & Laryng. **43**:1147 (Dec.) 1934.

31. Moersch, H. J., and Camp, D. J.: Diffuse Spasm of the Lower Part of the Esophagus, Ann. Otol., Rhin. & Laryng. **43**:1165 (Dec.) 1934.

32. Mosher, H. P.: Notes on Esophagus Cases, Ann. Otol., Rhin. & Laryng **43**:1154 (Dec.) 1934.

dilatation of esophageal stenosis. In both cases the stricture was caused by caustics. In both there was a large pouch at the upper end of the esophagus, and the opening of the esophagus was excentric. In one of the cases the stomach had been pulled into the chest half way to the arch of the aorta. Both patients were treated by fluoroscopic dilatation with a metal-tipped bougie. Mosher expressed the opinion that the fundamental cause of cardiospasm is fibrosis caused by infection of the lower end of the esophagus. In addition, there is also a twist of the terminal portion. It has been shown that chronic infection of the lower end of the esophagus often accompanies disease of the gallbladder or liver.

In reviewing a series of forty-three cases of diverticula of the thoracic portion of the esophagus, Vinson [33] was impressed with the difficulty of evaluating symptoms and of associating them with the lesion in the esophagus. Thirteen of the patients had had previous respiratory infection, and three, tuberculosis. In eight there had been injuries to the thorax. In eighteen the diverticula were situated in the middle third of the esophagus, while in the remainder they were in the lower third. Esophagoscopic examinations were made in a number of the cases. In some patients the opening of the sac could be demonstrated, while in others it could not be identified, but secretion could be seen pouring from the sac into the esophagus. In the majority the esophagus appeared normal. In one patient the sac had ruptured into the left bronchus, producing an esophagobronchial fistula.

Benign Tumors of the Esophagus.—After observing a man, aged 45, who complained of symptoms of esophageal stenosis for nearly four years, during which time several roentgen studies were carried out, a positive diagnosis was finally made by esophagoscopy. Hicquet and Jourdain [34] made a direct examination, which disclosed an annular, ulcerated tumor at the lower end of the esophagus. The histologic diagnosis on the basis of tissue removed esophagoscopically was adenoma. Roentgen examination and esophagoscopy two years later revealed no change in the caliber of the stenosis, but the ulceration had healed.

In Kramer's [35] patient, a sessile fibrolipoma was removed from the right arytenoid cartilage and right pyriform fossa. Two years later a pedunculated fibrolipoma 4½ inches (11.4 cm.) long was found arising from the left side of the cricoid cartilage. According to Kramer,

33. Vinson, P. P.: Diverticula of the Thoracic Portion of the Esophagus, Arch. Otolaryng. **19**:508 (April) 1934.

34. Hicquet and Jourdain: A Case of Adenoma of the Esophagus, Scalpel **86**:231, 1934.

35. Kramer, R.: Multiple Fibrolipomata of the Hypopharynx and Esophagus, Ann. Otol., Rhin. & Laryng. **43**:881 (Sept.) 1934.

these tumors are interesting, first, because of the symptomatology, which may manifest itself either by sudden death from asphyxia or by practically complete absence of disturbances, except irritation of the throat, which is often considered neurotic; and second, by the bizarre appearance of an oyster-like or fleshy mass appearing in the fore part of the mouth and suddenly disappearing, leaving no trace of its presence.

Carcinoma of the Esophagus.—In his experience in more than three hundred cases of carcinoma of the esophagus, Souttar [36] found that roentgen examination surpassed all the methods employed in establishing a diagnosis. In his opinion, esophagoscopy is more useful in discovering the character of the growth and the possibilities of treatment than in the diagnosis of malignant stricture. The information obtained may be of great value, but esophagoscopy should not be undertaken lightly; only experts should employ this procedure, as the use of the instrument is by no means free from danger. This is especially important in a study of carcinoma, as the esophagus may be deeply ulcerated.

In reporting cases of carcinoma of the esophagus, Bernstein [37] emphasized the need of early diagnosis if results are to be secured in the surgical treatment. The employment of roentgen examination and esophagoscopy gives conclusive diagnostic results in all cases. Unfortunately, however, there may be delay in utilizing these diagnostic means early, because the symptoms are often misinterpreted. The symptoms of dysphagia should always be carefully investigated in all cases, and a diagnosis of globus hystericus should be made only after all other possibilities have been ruled out.

Keefer [38] directed attention to the fact that carcinoma of the esophagus may disguise itself in the form of a chronic pleural or pulmonary infection. Seventeen cases of carcinoma of the esophagus are recorded in which pleural or pulmonary complications were the outstanding features of the disease. Pleural and pulmonary complications of carcinoma of the esophagus result from perforation of the growth into the trachea, bronchi, lungs or pleurae, from obstruction of the air passages, the aspiration of food or the perforation of the lung by a metastastic necrotic process in a lymph node. The symptoms and signs caused by these complications may completely dominate the clinical picture and overshadow symptoms of the esophageal lesion.

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36. Souttar, H. S.: Cancer of the Oesophagus, Brit. M. J. **2**:797 (Nov. 3) 1934.

37. Bernstein, M.: Early Diagnosis of Carcinoma of the Esophagus, M. Rec. **139**:79, 1934.

38. Keefer, C. S.: The Pleural and Pulmonary Complications of Carcinoma of the Esophagus, Ann. Int. Med. **8**:72 (July) 1934.

The conditions that are to be differentiated from these complications are discussed.

In discussing the part played by bronchoscopy and esophagoscopy in disease, Souper [39] commented on the lamentably small number of patients who are referred for bronchoscopic investigation. In an analysis of his statistics it was found that almost 90 per cent of the entire number of patients examined were referred for esophagoscopic examination. In summing up his impressions concerning malignant disease of the esophagus, he agreed that in far too many cases the condition is labeled neurotic or functional and that the cases are not investigated properly until the tumor has reached a size which renders any method of treatment almost impossible. If dysphagia occurs in a patient of an age when cancer is likely to develop, whether the symptoms are referred to any particular area of the esophagus or not, he should be referred without delay for the fullest investigation, an investigation which must include esophagoscopy. Attention is also directed to the interpretation of negative findings in roentgen examination. Early esophageal lesions may give negative findings. Furthermore, roentgen evidence suggesting cardiospasm in a patient over middle age whose symptoms are of recent development should not lead to the belief that the case is one of pure cardiospasm. These patients should be submitted to esophagoscopy to rule out the possibility of malignant disease. No case of this kind should be investigated by a bougie.

The occurrence of infection of the esophagus during an attack of Malta fever, with subsequent development of marked cicatricial stenosis, was reported by Malan.[40] The patient, a farmer, aged 18, who handled goats, contracted Malta fever. During the early stages he complained of intense pain in the stomach, with a burning sensation along the esophagus. There were periods of severe vomiting. The attack of fever was severe and continuous for practically three months. Convalescence was slow, owing to extreme emaciation and anemia. At this time it was noted that there were dysphagia and dysphonia, with pain in the region of the upper part of the esophagus. By mirror laryngoscopy the left side of the larynx was found fixed. Roentgen study of the esophagus revealed marked stenosis at the upper third of the thoracic esophagus, with dilatation proximal to the stricture. At esophagoscopy a tight stricture about 5 cm. in length was found at about the level of the aortic arch. The scarring was extensive but apparently superficial. The stricture responded rapidly to dilatation.

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39. Souper, H. R.: Bronchoscopy and Esophagoscopy in Diagnosis and Treatment, Brit. M. J. **1**:592, 1934.

40. Malan, A.: Esophageal Stenosis Complicating Malta Fever (Brucella), Arch. ital. per le mal. d. trachea-bronchi, esophago **12**:79 (April) 1934.

Gastroscopy.—Benedict [41] discussed the technic of gastroscopy with the new Wolf-Schindler flexible gastroscope. He was impressed that this method of examination is easy and harmless and expressed the opinion that from the standpoint of information secured it is a profitable procedure, and that it is a valuable adjunct to roentgenography in the diagnosis of gastric disorders. As the gastroscopic findings in ulceration and new growths are dependent on direct inspection of the mucous membrane of the stomach, they are particularly valuable. Benedict stated, however, that the greatest field of usefulness of gastroscopy lies in the investigation of gastritis. His views were based on a study of seventy-five patients.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES

In a report covering fifteen selected cases of a foreign body in the air and food passages which exemplified certain problems in treatment, Miller [42] directed attention to some observations that he had made. Special emphasis was placed on the difficulties of esophagoscopy and the fact that it is more dangerous and difficult than bronchoscopy, especially in infants and in the aged. He has made it a rule in cases of an esophageal foreign body to tilt the table in the Trendelenburg position. A brief note of the technic employed in each case is recorded.

Seydell [43] discussed the symptomatology of a foreign body in the air and food passages and enumerated the indications and contraindications for diagnostic bronchoscopy and esophagoscopy. He stated that the physician must be foreign body-minded, and that if a careful history was taken few foreign bodies in the air and food passages would be overlooked. The initial symptoms which follow the aspiration or swallowing of a foreign body may be slight or even absent, giving rise to a symptomless interval. After a diagnosis of a foreign body has been made removal should be proceeded with. Few foreign bodies are expelled spontaneously.

In a report of a series of cases of a foreign body in the upper air and food passages observed between 1912 and 1932, Noury [44] recorded in detail the methods of extraction with endoscopic tubes. The foreign

41. Benedict, E. D.: Examination of the Stomach by Means of the Flexible Gastroscope: A Preliminary Report, New England J. Med. **210**:669 (March 29) 1934.

42. Miller, J. W.: Foreign Bodies in the Air and Food Passages; a Plea for Early, Deliberate and Careful Extraction, Laryngoscope **44**:642 (Aug.) 1934.

43. Seydell, E. M.: Practical Considerations of Bronchoscopy and Esophagoscopy, J. Kansas M. Soc. **35**:451 (Dec.) 1934.

44. Noury, Z.: Bronchoesophagoscopy for Foreign Bodies of the Upper Air and Food Passages, Bronchoscop., oesophagoscop. et gastroscop. **4**:315 (Oct.) 1934.

bodies removed from the esophagus numbered twenty-six, while ten were removed from the larynx and bronchi. He preferred the Bruening instruments. Esophagoscopy was performed with the patient in the sitting position. He expressed the belief that the prone position seems to be too fatiguing to the patients.

In presenting a series of forty-one cases of a foreign body in the air and food passages removed by peroral endoscopy, Gatewood and Hughes [45] briefly discussed the symptoms of a foreign body lodged in the esophagus, larynx, trachea and bronchi. They emphasized the seriousness of organic foreign bodies in the air passages of children and the problems presented by these cases. To be successfully handled, they require the greatest degree of skill and judgment available, combined with experienced team-work and adequate equipment.

Bernard and Soulas [46] directed attention to the differences in the evolution and prognosis of suppuration resulting from an endobronchial foreign body, as compared with the non-foreign body forms. Three cases are presented in detail. In one, in which the foreign body was a tooth, it was coughed up, while in the remaining two, it was removed. In all three cases definite bronchiectasis was present. This cleared up promptly after a few bronchoscopic treatments. In the experience of these authors, pulmonary abscess is rarely a sequel of the presence of a foreign body; their observations would seem to corroborate the view that bronchiectasis more often develops. The prognosis is dependent in part on whether or not bronchoscopic treatment is continued after removal of the foreign body.

The unusual features of the case of a foreign body in the trachea, reported by Tucker,[47] are noteworthy in that the foreign body, one half of a wooden tongue depressor, remained in the tracheobronchial tree for a period of thirteen years, during which it was freely movable and produced only a slight inflammatory reaction of the mucous membrane. It was coughed into the subglottic part of the larynx and became impacted, producing sufficient reaction to cause marked stenosis of the larynx. Following removal of the foreign body the patient was left with chronic stenosis of the subglottic part of the larynx, which could be ascribed only to the trauma of the foreign body. The chronic stenosis responded to rest, following low tracheotomy and repeated direct laryngoscopic bougienage.

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45. Gatewood, M.D., and Hughes, T. E.: Bronchoscopy and Esophagoscopy; Report of Cases, Virginia M. Monthly **60**:709 (March) 1934.

46. Bernard, L., and Soulas, A.: Unsuspected Endobronchial Foreign Bodies and the Bronchoscopic Treatment of the Resulting Bronchopulmonary Suppurations, Bronchoscop., oesophagoscop. et gastroscop. **4**:277 (Oct.) 1934.

47. Tucker, G.: Wooden Tongue Depressor in the Trachea Thirteen Years: Chronic Laryngeal Stenosis, Ann. Otol., Rhin. & Laryng. **43**:1124 (Dec.) 1934.

In discussing the problems caused by nuts as foreign bodies in the air passages, Richards and Walker [48] presented their observations on twenty-six patients who had been treated by bronchoscopy. The importance of inquiring into the possibility of aspiration in taking the history in any case of unexplained pulmonary pathologic condition is stressed. The characteristic physical signs are enumerated and are shown in a majority of instances to be typical in this condition. The roentgen findings are also summarized, the typical findings illustrated and variations explained. The question of anesthesia is discussed, and the merits of no anesthesia and of avertin and ether anesthesia are presented. The bronchoscopic findings are analyzed, and the problem of fragmentation of the nuts is dealt with in detail. Complications are considered and estimated according to the immediate and the later effects, together with the measures necessary to combat them, such as tracheotomy or intubation. The end-results show permanent recovery in twenty-five cases and death in one.

An experimental study to determine the reaction of pulmonary tissue in the presence of certain foreign bodies was reported by Hara.[49] Rabbits were employed in the experiments. The materials used for introduction in the bronchi consisted of ten different organic foreign bodies; these were placed in position with the forceps, under visual guidance, through a specially constructed bronchoscope. The animals were killed at the end of the first, second, third and fourth day. In summarizing the findings, it was noted that the reaction in the lung increased with each subsequent day until the pulmonary parenchyma lost its normal architecture. The area of exudate formed was always limited to the immediate neighborhood of the larger bronchi and extended outward. The rapidity with which this expansion took place appeared to be in direct proportion to the degree of irritation. Secondary changes in the lungs were always localized, never lobar, pneumonitis. These occurred on the third or fourth day in lungs containing the most irritating foreign bodies. Vascular changes were uniformly those of obliterating arteritis, with foreign bodies causing marked reactions. This proliferative change in the endothelial lining of the blood vessels brought on successively infarction and focal necrosis of the pulmonary parenchyma. Frank capillary hemorrhage in the alveolar wall and, to some extent, hemorrhage in the alveoli, were noted in lungs containing popcorn, bark of the eucalyptus tree, seeds of watermelon, grapefruit and orange and, particularly, peanuts. It was interesting to note that similar changes were noted in the opposite lung, which contained no foreign body.

48. Richards, L. G., and Walker, J.: Nuts as Bronchial Foreign Bodies, New England J. Med. **211**:653 (Oct. 11) 1934.

49. Hara, H. J.: Organic Foreign Bodies in the Bronchi, Arch. Otolaryng. **20**:549 (Oct.) 1934.

An interesting case of obstructive atelectasis due to retention of secretion was reported by Tucker and Wolferth.[50] A head of timothy had been removed bronchoscopically in the case of a boy, aged 13 years. A large quantity of secretion was released from the obstructed area of lung beyond the foreign body. The intern gave morphine to quiet the patient's cough, with the result that complete atelectasis of the entire lung developed. Bronchoscopic removal of the obstructing secretion was followed by prompt clearing up of the atelectasis. No further sedative was given, and the patient made a prompt recovery.

What would appear to be a hazardous procedure was practiced by Equen,[51] who removed from the esophagus a bobbin which had been swallowed about two and a half years previously. All the endoscopic manipulations, including cutting through the scar tissue, were carried out under biplane fluoroscopic guidance. Equen emphasized the importance of immediate and careful investigation in cases of a suspected foreign body. Prompt removal of foreign bodies obviously prevents the development of serious complications and obviates the need of employing measures that are not without danger.

Regurgitation of a foreign body from the stomach and its subsequent lodgment in the esophagus are uncommon. In the case reported by Roy,[52] an open safety-pin which had lodged in the thoracic portion of the esophagus could not be found at esophagoscopy. By fluoroscopic localization the pin was found to be in the stomach. The following day fluoroscopic examination was carried out and the pin observed in the esophagus, with the point and keeper downward. It was learned that the patient had had a violent attack of vomiting during the night. Removal by esophagoscopy was a relatively simple problem, and the patient made a complete recovery.

Arens and Stewart [53] reported the occurrence of pneumopericardium in an 8 month old infant who had swallowed a safety-pin. There was no history of the presence of a foreign body previously. The symptoms of dysphagia, loss of weight and occasional blood in the stools were of five months' duration. The foreign body was observed on roentgen examination. The observations at autopsy indicated that the pin had perforated the anterior wall of the esophagus and had entered the peri-

50. Tucker, G., and Wolferth, C. C.: Obstructive Atelectasis Following Removal of Foreign Body from the Lung Due to Viscid Secretion and Inhibition of Cough by Morphin, Ann. Otol., Rhin. & Laryng. **43**:1202 (Dec.) 1934.

51. Equen, M.: Sharp Dissection Through an Esophagoscope: Removal Under Biplane Fluoroscope of Bobbin in Esophagus, J. A. M. A. **103**:1845 (Dec. 15) 1934.

52. Roy, J. M.: Open Safety Pin in the Stomach Regurgitated into the Esophagus and Removed by Esophagoscopy, Canad. M. A. J. **30**:646 (June) 1934.

53. Arens, R. A., and Stewart, E.: Pneumopericardium Following a Foreign Body in the Esophagus, Radiology **22**:334 (March) 1934.

cardium. A well formed fistula connected the pericardial cavity and the esophagus. Reported cases of pneumopericardium following the presence of a foreign body in the esophagus are rare; only four cases were noted in the literature by these authors. In three of these the patients were adults; in the remaining case the patient was an infant. It is probable that the condition is not so rare as it appears, since the symptoms and signs are indefinite and often misleading and are usually diagnosed only from the roentgenologic findings or the observations at autopsy. In this case the diagnosis was made from the roentgenograms and was confirmed by the observations at autopsy.

In three cases of punctured wounds of the esophagus caused by a fragment of bone, Wright [54] was able to remove a sharp pointed fragment of bone in two cases; in the third, perforation of the esophageal wall was found, but no foreign body could be identified. One of the cases terminated fatally; in the second case the patient recovered following gastrostomy and external drainage of the collection of pus at the root of the neck. In the third case, in which no foreign body was found, the patient recovered promptly. Wright expressed the opinion that gastrostomy is indicated as soon as there is evidence of the passage of food through a perforation in the esophageal wall. When the presence of a sharp fragment of bone is likely, esophagoscopy should be carried out at the earliest possible moment, in spite of negative findings on roentgen examination. Suppurative complications may follow a puncture wound of the esophagus in spite of early removal of the foreign body, but such complications are not necessarily fatal. Local tenderness on pressure over the trachea is a sign of considerable importance in such a case. Blind passage of instruments should be particularly avoided, as this is likely to force the foreign body deeper into the tissues.

In Heatly's [55] case, a toothpick which had perforated the wall of the upper part of the esophagus was removed esophagoscopically within five hours after it had been swallowed. In spite of this, periesophageal infection occurred, and external drainage was resorted to. The patient made a complete recovery.

In the case reported by Butler,[56] there was instrumental trauma to the esophagus at the cricopharyngeal level. This was recognized at the time, and the esophagus was exposed through an external incision.

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54. Wright, A. J.: Punctured Wounds of the Oesophagus, J. Laryng. & Otol. **49**:175 (March) 1934.

55. Heatly, C. A.: Minute Perforation of the Cervical Esophagus; Fulminating Descending Infection; Mediastinitis; External Operation; Recovery, Ann. Otol., Rhin. & Laryng. **43**:873 (Sept.) 1934.

56. Butler, E. F.: Putrid Mediastinal Abscess with Spirochaetal Infection: Report of a Case, Ann. Otol., Rhin. & Laryng. **43**:878 (Sept.) 1934.

A small tear found in the esophageal wall was packed off, but in spite of this mediastinal infection occurred. Large spirochetes were found in abundance in the exudate. Excellent results were secured following irrigation of the abscess cavity with an aqueous solution of neoarsphenamine.

To overcome certain difficulties in the localization of foreign bodies in the upper part of the abdomen in relation to the stomach, Tucker [57] passed a rubber feeding tube and had roentgen films made with the tube in situ, with the patient in the position required for gastroscopy. The advantages claimed for this method are that it shows the location of the foreign body with reference to the tube that is passed into the stomach. The tube stays in the lower part of the esophagus and the cardiac end of the stomach, where the gastroscope is to be introduced. The rubber tube is withdrawn, and gastroscopic removal can be proceeded with immediately. The contents of the stomach can be aspirated and air introduced, if desired, before the tube is withdrawn.

In an article by Solbrig [58] on the limitations of endobronchial therapy, no mention was made of bronchoscopy for disease; cases of a foreign body only were considered. Though this author advocated bronchoscopy in these cases, he cited a high percentage of failures, and in reading of most of them one is impressed by the fact that bronchoscopic methods for one reason or another did not have a fair trial, in fact, in quite a number of cases no trial at all. It seems deplorable that thoracotomy was done in six of fourteen cases of an endobronchial foreign body without any attempt at bronchoscopy. In nine of the cases the patients recovered after pneumotomy; five patients died. In one case the foreign body was not removed at thoracotomy because it could not be found. Some of the statements in regard to bronchoscopy for removal of a foreign body will be questioned by many brochoscopists. Some examples may be cited: It is stated that mucus, pus and blood may present insurmountable difficulties in cases of children, and also that granulations and "the marked tendency to severe hemorrhage" render the prospects not good in such cases. In dealing with granulations "a procedure in stages in rapidly successive sittings to prevent regrowth" is advised in adults, but is stated as being impossible in children. In cases of "foreign bodies lying in secondarily formed cavities, whether bronchiectasis or abscesses," bronchscopy is regarded as futile. Pneumonotomy was advised in the illustrative case cited and was fatal. In the case of an adult with a radium needle deep in the lower lobe it is said that "extrac-

57. Tucker, G.: A Method of Roentgen Localization of Foreign Bodies in the Stomach Prior to Gastroscopic Removal, J. A. M. A. **103**:1440 (Nov. 10) 1934.

58. Solbrig, H.: Limitations of Endobronchial Therapy, Monatschr. f. Ohrenh. **68**:338 (April) 1934.

tion was impossible as between the needle and the lowest edge of the 7 mm. bronchoscopic tube there was a gap of 1 to 1½ cm." Pneumonotomy was done, which was followed by death "from cardiac failure" in five hours. Costophrenic bronchoscopy and the methods of Jackson for the removal of pins, needles and other slender objects from the periphery of the lung were not mentioned. In one case cited from the literature two attempts were made to reach a pin at the bottom of the lower lobe of the right lung with a tube 34 cm. in length. As this length proved insufficient, tracheotomy was done. The tube was then found to be long enough to reach, but the pin could not be removed, and pneumonotomy was done.

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Abstracts from Current Literature

Ear

DEAFNESS: FREQUENCY, CLASSIFICATION AND SOME OF THE COMMON CAUSES. STANWOOD E. FISHER, Maine M. J. **24**:11 (Jan.) 1934.

The prevention of deafness is concerned with the detection of impaired hearing in its earliest stages. Even in childhood this condition involves economic loss by necessitating repetition of grades in school and the maintenance of public institutions for the extremely deaf.

Of 2,750,000 men examined for the draft, 32,000, or 1.5 per cent, were found to have aural defects. About 10 per cent of school children suffer from some defect in hearing. The United States census of 1930 reports 57,084 deaf-mutes who either were born deaf or supposedly lost their hearing before the age of 8 years.

Elimination of causes and early detection of cases, with prompt use of therapeutic measures are most important in prevention, because in 75 per cent of cases the condition can be arrested or benefited if seen early. Causes comprise: acute contagious diseases, enlarged tonsils and adenoids, infections of the upper respiratory tract, abnormalities of the nasal passages and sinuses, the marriage of persons with a family history of deaf-mutism or otosclerosis, industrial and other noises, suppuration of the ear, syphilis, unsanitary swimming pools and detrimental drugs, such as quinine, salicylates and arsenicals.

A simple test calls for the hearing of an ordinary Ingersoll watch at a distance of 48 inches (1.2 meters) and of the whispered voice at 20 feet (6.09 meters). In examining large groups, the audiometer may be used by school nurses to screen out the pupils with impaired hearing in a given group.

Most children complain of deafness only after they are well along in school age. However, those affected repeat more grades than other children. Reticence and sensitiveness often result from the poor hearing. Progressive deafness due to fixation of the conductive apparatus and deafness produced by degeneration of the eighth nerve are almost never found in children. Three fifths of the cases of deaf-mutism are accounted for by heredity and consanguineous marriages. Acquired deafness is found in less than half the children in public institutions for the deaf. This form is usually of the tubotympanic type, is not so severe as the congenital form, is intermittent and is traceable to unfavorable climate inducing frequent colds, with the involvement of sinuses. Undernourishment may have some etiologic relationship to deafness.

Deafness of middle age and senile deafness are also discussed.

GRAVES, New York. [AM. J. DIS. CHILD.]

THE "ACUTE EAR" IN GENERAL PRACTICE. DOUGLAS GUTHRIE, Brit. M. J. **1**:4 (Jan. 6) 1934.

The author considers the causes and clinical course of acute otitis media, the atypical varieties of this condition, the appearance of the drum, the appearance of the patient, the indications for incision and the complications, including mastoiditis, along more or less well known and conventional lines.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

HAEMATURIA IN CHILDHOOD ASSOCIATED WITH ACUTE MASTOIDITIS. LAURA L. BATEMAN, Brit. M. J. **1**:60 (Jan. 13) 1934.

A case of acute mastoiditis accompanied by hematuria in a child 6 years of age, is reported. An operation was performed on the mastoid, and within five days the hematuria had disappeared. The conclusion is that hematuria associated with mastoiditis indicates early operation.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

THE OTOSCLEROSIS PROBLEM: INCLUDING REPORTS OF TWO CASES EXAMINED PATHOLOGICALLY. A. A. GRAY, J. Laryng. & Otol. **49**:629 (Oct.) 1934.

Gray reports two cases of otosclerosis examined pathologically. One of them was also examined clinically. As a result of his observations and from other known facts he concludes that the essential causative factor of otosclerosis is a gradually increasing defect in the vasomotor mechanism that governs the nutrition of the structures of the organ of hearing as a whole. The axon reflexes are, of course, included in this vasomotor mechanism, and the stimulus that excites the vasomotor mechanism is sound and sound alone. Consequently, the vestibular apparatus and the semicircular canals are unaffected in otosclerosis. There is no evidence of defect in any of the endocrine glands or in their secretions in otosclerosis. Neither is there evidence of any defect in the bone metabolism of the body. On the contrary, the subjects of otosclerosis are, apart from their deafness, perfectly normal persons with average health. The deafness of a person with otosclerosis bears little relationship to the extent of the disease in the bone. The deafness may be severe when the stapes is hardly fixed at all. The severity of the tinnitus bears no relationship to the extent of the disease in the bone. The extent of the change in the bone bears little relationship to the duration of the disease. The extent of the changes in the bone appears to depend on the age of the patient at the onset of the disease. The earlier the age at which the otosclerosis begins, the more extensive will the lesion of the bone become. In otosclerosis the deafness is to a large extent functional and results from an insufficient supply of blood to all the nerve structures concerned in the perception of sound. The preponderance of women among the subjects of otosclerosis is a result of the greater instability of their vasomotor system and the greater frequency of its exposure to disturbances. The changes in the bone show bilateral symmetry even to minute details. This symmetrical distribution is readily explained by the author's view of the causative factor of otosclerosis. The vasomotor nerves governing the nutrition of the organs of hearing are anatomically symmetrical, as are other nerve structures in the body. If, therefore, structural changes occur as a result of defective functioning of those nerves, such changes will be bilaterally symmetrical in their distribution.

EDITOR'S ABSTRACT.

AURAL SYMPTOMS IN A PERSON STRUCK BY LIGHTNING. S. COMES, Arch. ital. di otol. **46**:241 (April) 1934.

Comes describes a man, aged 43, who remained unconscious for two days after the electric shock caused by his being struck by lightning and on awakening presented loss of hearing in both ears. A few days later pus was found in the left auditory canal. The patient had not complained heretofore of pains in the ear. Otoscopic examination revealed that shortly previous to the trauma there was purulent otitis media of the right ear with tympanic perforation. It is possible that the tympanic perforation in the left ear also preceded the electric trauma or, as in the right, had been already cleared up. The inflammatory processes were secondary, thus explaining how the otorrhea occurred without any pain. Functional examination revealed bilateral deafness in which the tympanic lesions were not capable of producing marked hypo-acusia. The disappearance of bone conduction, the total deafness of the left ear to the tuning fork and the disappearance of con-

duction of high and low sounds in the right ear point to a lesion of the apparatus of perception. The patient showed nervous manifestations, indicating that the electric shock had affected the central nervous system and in turn the auditory nerve. The concomitant neurotic factors affecting the auditory nerve were nuclear phenomena induced at a distance by vasomotor lesions.

EDITOR'S ABSTRACT.

THE CAUSES OF DEAF-MUTISM. SUNÉ Y MEDAN, Paidoterapia **12**:1099 (March) 1934.

The author gives special attention to the latent otitis of the sucking baby. He insists above all that hereditary deaf-mutism is as frequent as acquired deaf-mutism, and Parrel admits that the number of persons with congenital deafness is greater than the number of those with acquired deafness.

Syphilis is an important cause of this condition. It may produce deafness before or after birth, at about the fifth year or, if late, from the seventeenth to the thirtieth years. If it supervenes before the child starts talking, he will remain a deaf-mute. The Wassermann reaction has only a relative informative value. Other congenital causes include a hereditary tendency, physical traumatism of the mother during the pregnancy, nervous defects and consanguinity. Deafness due to consanguinity is as high as 10 per cent, according to some authors.

Causes of acquired deafness include traumatism to the head during early infancy, cerebrospinal meningitis and latent otitis media of the suckling. Latent otitis occurs most frequently from the sixth to the ninth month. It is best prevented by careful examination of the ears of all infants and the early treatment of otitis; by the prevention of marriages between blood relatives, especially when there is deafness in the family of one of the parties, and by repeated careful examination of the ears of all infants with fever.

HERRON, Spokane, Wash. [AM. J. DIS. CHILD.]

NEURITIS OF THE AUDITORY NERVE? M. KRASSNIG, Monatschr. f. Ohrenh. **68**:555 (May) 1934.

Krassnig has observed a number of visible acute extratympanal disturbances of the ear. All showed a similarity in functional deficiency and course, so that a uniform etiologic factor could be considered for all. All of the six cases considered were analogous in that inflammation of the inner ear tube similar to Ménière's disease evolved suddenly, but every sign of acute inflammation of the tube was absent. The condition occurred in healthy persons and, with one exception, in completely normal organs of hearing. All of the cases observed by the author so far have been one-sided and have involved the cochlea as well as the vestibule. The majority of the patients, after weeks or months, showed a distinct, often extensive but never complete restoration of the disturbed function.

Two points differentiate this condition from the usual Ménière's symptom complex: 1. The Ménière state affects the middle ear and recurs frequently. The preexisting disturbance of hearing becomes worse during an attack but passes with the attack. Vertigo takes a prominent place in the picture. The author's cases, on the contrary, have so far involved a single disturbance and the hearing has not entirely returned to normal. 2. In addition to disorders of the middle ear, general conditions, such as syphilis and hypertonia, cause the Ménière state. In such cases one is dealing with a symptomatic Ménière disease. The questions of the site and nature of the pathologic process are important. These cannot be answered with certainty, but the author suggests that in some instances a sort of neuritis of the auditory nerve exists which should be removed from the large group of disorders resembling Ménière's disease. The treatment which has given the best results so far has consisted of injections of pilocarpine and the administration of tea.

EDITOR'S ABSTRACT.

HEARING PERCEPTION IN JUDGMENT OF OPERATING MACHINES. A. A. GLADKOFF, Monatschr. f. Ohrenh. **68**:560 (May) 1934.

Gladkoff carried out investigations on the ability of normal persons to hear and project correctly abnormal sounds emanating from several different kinds of machines in operation. The protocols for some are given in detail. The results varied considerably in different persons and with sounds of different nature. As a result, he believes that it is necessary that the term "machine hearing" should receive the same consideration as the term "musical hearing," and the degree of hearing ability of the technical engineer should be determined in this respect. It is especially important that this testing be compulsory, if not by laboratory means at least on the operating machine, so that the hearing of the examinee can be determined according to the nature of the abnormal sound. He feels that it would be desirable also to establish a scientific laboratory for the study of the sound waves of operating machines and of the ability of people to orient the different accessory sound effects. Improvement in the knowledge of these effects, he believes, should decrease the number of injuries and accidents, improve the quality of production and save money.

EDITOR'S ABSTRACT.

AUDITORY PROJECTION OF DIRECTION OF AUTOMOBILE SIGNALS IN CITY NOISE AND ON COUNTRY ROADS. R. PERWITZSCHKY and H. BITTERAUF, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:463 (April 6) 1934.

One of the new rules affecting drivers of motor vehicles in Germany requires that such drivers be able to determine the direction from which warning horns come. Since it is not known on what scientific studies this ruling was based it is assumed that it is purely bureaucratic in origin. Perwitzschky and Bitterauf observed the projection of hearing in a number of persons with normal hearing in various places in the city and on rural roads. It was found that on the average in city traffic 50.2 per cent of all signals were placed correctly when the subject remained in the same spot. Twenty-three per cent were falsely projected, and in the case of 26.6 per cent the direction was indeterminate. These results became worse when the subject was himself placed in a moving vehicle. To explain this poor accuracy, it has been suggested that the buildings in the city reflect the sound waves and are therefore responsible. To test the truth of this contention, observations were made on country roads poor in reflectors. When the observer stood in a lane surrounded by high trees the false projection was 41.1 per cent. In a free stretch of country road only 25 per cent of all signals were falsely heard. In an open field the false projection fell to 8 per cent. The authors feel, therefore, that these observations corroborate the belief that most false projections are due to reflection of the sound waves. Further observations were conducted on a person with one-sided deafness, with such poor results that correct projection of the sound waves occurred rarely. The authors conclude from these observations that it is questionable whether the recent law in regard to correct localization of sound is of practical importance.

EDITOR'S ABSTRACT.

ROENTGEN DIAGNOSIS OF DISEASES OF THE PETROUS BONE. J. KRÖHNKE and F. KUHLMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:526 (May 15) 1934.

Kröhnke and Kuhlmann report several roentgenologic observations made by a modification of Stenver's technic. The principle of Stenver's method lies in the projection of an unshortened pyramid on a portion of bone which has little specific structure. The authors, however, took the pictures with the patient sitting rather than with him lying with the head flat as in Stenver's original procedure. Except for this change Stenver's technic was strictly followed. With this method the authors were able to draw extensive conclusions on the nature and extension of some rare cases of disease of the petrous bone. In a girl, aged 7, the roentgenogram showed extensive pneumatization of the left mastoid process without special

changes in the inner ear. In the apex of the pyramid was a vertically placed oval light area (cyst) which was surrounded by a thickened wall. With this exception the bony structure was normal. In the right mastoid process no cellular structure was visible. Two centimeters from the apex of the pyramid the contour of the pyramid was peculiarly narrow, thin and somewhat interrupted at circumscribed places. In the adjacent bone the usual structure was no longer visible. Thus roentgenologic observation confirmed the clinical diagnosis of extradural abscess with involvement of the pyramidal apex (Gradenigo's symptom complex). In another patient, aged 3½ years, the frontal view showed larger and smaller bone defects, which were outlined with rather sharp walls. The larger defects were in the region of the upper frontal bone and in portions of bone near the mastoid. In the region of the concha of the ear a somewhat thick, tongue-like shadow was visible. Finally, the authors describe the roentgenologic observations in a patient with long-existing Paget's disease. The chief changes of the osteitis deformans were localized in the tibias, the lumbar vertebrae and the skull. Pyramidal abnormalities were found bilaterally.

EDITOR'S ABSTRACT.

FATAL COMPLICATION FROM A COMPLETELY OPENED AND HEALED EAR. F. OTTEN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **25**:117, 1934.

Otten reports the case of a laborer, aged 31, in whom purulent meningitis proved fatal. Necropsy revealed the source of infection to be a tonsillar inflammation which reached the meninges by way of an old operative passage. Histologic examination showed two paths by which the infection reached the meninges: one through the injured labyrinth to the inner ear passage and one made by an earlier operative fissure to the dura. The latter appeared to be the more important. The earlier operative procedure had been performed about nine years previous to the fatal illness, and there had been complete healing. The author feels that in no way can responsibility for the meningitis be placed on the procedure of complete opening, nor does it shake his opinion of the value of complete opening as a preventive measure against complications developing from the ear.

EDITOR'S ABSTRACT.

CENTRAL DISORDERS OF HEARING CAUSED BY ABSCESS OF THE TEMPORAL LOBE. L. M. KOMPANEYTS, Vrach. delo **16**:383, 1933.

The author reports five cases of abscess of the temporal lobe, and gives the results of an investigation of the auditory changes. Studies were made of the auditory changes on the side opposite that on which the lesion was located, since it is known that most fibers of the auditory nerve decussate, and because too many complicating factors are present on the same side. The results obtained were: (1) shortening of the bone conduction with the low tone tuning forks Ç and A and (2) diminished perception of sound for the lower range tones, i. e., either by air or by bone conduction. The author's conclusions are:

1. For normal qualitative perception of sound, the integrity of both auditory and subcortical centers is necessary.

2. With involvement of the auditory center and subcortical tracts on one side, there is observed a diminution in the perception of sound, particularly for the lower range of the auditory scale. This would suggest that the decussated fibers of the auditory nerve are primarily fibers for the conduction of the low pitched tones, i. e., that there is a differentiated mechanism for the perception of sound.

3. The shortened bone conduction in cases of abscess of the temporal lobe would tend to show that the perception of sound through bone conduction depends not only on the condition of the peripheral nerve endings but on that of the cerebral cortex as well.

BODER, Chicago. [AM. J. DIS. CHILD.]

Pharynx

LUNG ABSCESS AFTER TONSILLECTOMY. F. T. RANSON and L. MCGOLRICK, Brit. M. J. **2**:1020 (Dec. 2) 1933.

The authors report a case of a pulmonary abscess following tonsillectomy, with operation and recovery. A brief discussion of the mode of production of such abscesses and their treatment follows.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

ADENOIDITIS, ADENOPATHY AND NASOPHARYNGEAL MASSAGE. G. VERGER, Arch. de méd. d. enf. **37**:21 (Jan.) 1934.

Forcible friction of the adenoid area by the finger was first practiced in 1926 by Robert Rendu of Lyon for inflammation of adenoid vegetation accompanied by regional adenopathy. In 1929, Gamaleia and Michon used the term "providential finger" in discussing the agent used in crushing these tissues. Although this method was first employed only in cases of prolonged adenoiditis, Verger now uses it in the acute stage, with equal success; the temperature, which may be elevated for many days in untreated patients, usually falls to normal on the day following massage. Verger believes that whether or not adenopathy accompanies acute adenoiditis, it is the latter which is responsible for the rise of temperature, or what is commonly known as glandular fever.

It is maintained that acute prolonged adenoiditis probably constitutes the chief source of continued hyperthermia in early childhood. The author reports seventeen cases observed, including cases of simple acute adenoiditis, several of the prolonged type and a number of instances in which cervical adenitis was a complication.

Before crushing the vegetation it is necessary to exclude otitis media; if this exists and is properly treated without causing a drop in the temperature, adenoiditis is suspected and is treated as has been described. Tuberculosis is excluded by a negative cutireaction and consideration of the child's environment; the elevation in temperature in the latter disease is also much more regular in its stride. Central pneumonia is difficult to differentiate at this time. It is assumed that the favorable action noted in this procedure follows the escape of minute quantities of pus collected between the lamellae of the adenoids.

AMESSE, Denver. [AM. J. DIS. CHILD.]

THE TONSIL PROBLEM. G. M. SUSIN, Prat. pediat. **11**:323 (Oct.) 1933.

Susin summarizes the various theories concerning the function of the tonsils and discusses the disparity in reported results of tonsillectomy. He concludes that a conservative attitude should be taken toward tonsillectomy and that even in cases of acute and chronic tonsillitis partial removal is preferable to complete extirpation. A good bibliography of the Italian literature on this subject is listed.

DOOLEY, Kent, Conn. [AM. J. DIS. CHILD.]

RHINOPHARYNGEAL DIPHTHERIA IN A NEW-BORN INFANT WITH SEVERE CONGENITAL SYPHILIS. N. L. BLOISE, Arch. de pediat. d. Uruguay **4**:276 (July) 1933.

The author reports a case of rhinopharyngeal diphtheria in a new-born infant with congenital syphilis as confirmed by a strongly positive Wassermann reaction of the blood of both the baby and the mother. Intensive antisyphilitic treatment was started at birth, but on the fourth day sanguinopurulent coryza appeared without the early cutaneous manifestations of syphilis. A few days later an extensive gray, false membrane suggestive of diphtheria was noticed in the pharynx. Cultures from the nose and throat were negative for spirochetes but revealed an abundant growth of Klebs II Löffler's bacillus. The culture from the mother's throat was likewise positive for the diphtheria bacillus. The baby died on the eleventh day of life, in spite of the intensive antisyphilitic and antidiphtheritic treatment.

The author points out the rarity of the condition and suggests that in all cases of coryza in syphilitic children resistant to the specific treatment nasal diphtheria should be ruled out.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

Larynx

VALLECULAR DYSPHAGIA. JOHN E. MCGIBBON and J. H. MATHER, Brit. M. J. **2**:1013 (Dec. 2) 1933.

Elucidation of the causes of dysphagia arising in the lower part of the pharynx has always presented difficulty. Gross neoplastic and ulcerative lesions can be recognized with the laryngoscope, but there remain a large number of undiagnosed conditions which have been termed "functional dysphagia" and "globus hystericus." The authors were stimulated by the report by Dr. R. R. Woods of a case in which the difficulty in swallowing was designated as vallecular dysphagia. They have observed fourteen cases of what they term primary vallecular dysphagia and a considerable number of cases which they classify as instances of secondary vallecular dysphagia.

The valleculae are two fossae situated in the anterior aspect of the lower part of the pharynx, between the pharyngeal portion of the tongue and the epiglottis. They are bounded anteriorly by the base of the tongue and laterally by ridges of mucous membrane—the pharyngo-epiglottic folds. They are separated from each other by the glosso-epiglottic fold. The floor of the valleculae lies immediately above and behind the hyoid bone. They vary much in depth in different persons. Their sensory nerve supply is derived from the glossopharyngeal and superior laryngeal nerves. Deglutition is a complicated reflex act, the afferent nerves being the second division of the fifth nerve, the ninth nerve and the pharyngeal branches of the superior laryngeal nerves. The efferent nerves are the third division of the fifth nerve and the seventh, ninth, tenth, eleventh and twelfth cranial nerves. The center is situated in the medulla and may be considered as a chain of associated centers, stimulation of one of which fires off the others in orderly sequence. The revised concept of the mechanism of swallowing is described, and the older explanation that the bolus of food is seized by the pharyngeal constrictors and forced into the esophagus, where it is passed on by peristalsis, is disproved; it seems that the passage of food through the lower part of the pharynx and upper part of the esophagus is accomplished by a combination of propulsion and negative pressure.

Careful screen examinations of the act of swallowing in more than one hundred normal men and women were made. In approximately 50 per cent of the cases the bolus of food was swallowed, leaving only a trace in the mouth and pharynx, none being seen to enter the valleculae. In the other 50 per cent, a little opaque material entered the valleculae but was evacuated almost immediately by their compression during the succeeding acts of swallowing.

Of the authors' fourteen cases of primary vallecular dysphagia, five were in females and nine in males. The usual complaint was discomfort on and after swallowing, which was referred to the region of the hyoid bone and had been present for from fourteen days to about two years. In some cases the discomfort was intermittent and occasionally persisted for some time after eating. In some cases the onset was insidious, and in others it was sudden. None of the patients complained of dyspnea or such marked symptoms as occurred in the case reported by Woods. Indirect laryngoscopic examination revealed the presence of food in the valleculae in only one case. Roentgenographic investigation showed that when patients were told to swallow, repeated attempts to do so were ineffectual in passing the bolus of food from the mouth into the pharynx. At each attempt a little food passed (1) into the valleculae, (2) into the right and left pyriform fossae and (3) into the pharynx and esophagus. General clinical examination in these cases revealed no systemic disease.

In what the authors have termed secondary vallecular dysphagia, the valleculae were filled in a similar manner, but this abnormality was due to a manifest lesion somewhere within the reflex arc or in the pharynx or esophagus itself. The

presenting symptoms were those of the primary lesion, and the vallecular filling and retention were discovered only by routine examination, the findings being similar to those in the primary form of dysphagia but less marked. This condition was observed in patients suffering from carcinoma of the esophagus, postcricoid carcinoma and pharyngeal pouch. It occurs in cases of bulbar paralysis, bilateral recurrent nerve paralysis, tabes with paralysis of the vagus, injury to the vagus and botulism following diphtheric paralysis, etc. It appears that it occurs only with lesions of relatively rapid evolution, since the neuromuscular apparatus seems to have the ability to accustom itself to abnormal conditions and has overcome the mechanical difficulties present.

The pathologic condition in primary vallecular dysphagia is doubtful, but from what is known of the pathologic process in the secondary form, it seems that there must be somewhere a failure in the neuromuscular mechanism of swallowing. The anterior group of muscles, consisting of the mylohyoid and the geniohyoid muscles, the posterior fibers of the genioglossus muscle and the anterior belly of the digastric muscle, are the most important. This group of muscles pulls the larynx upward and forward behind the base of the tongue and also pulls the larynx forward away from the posterior pharyngeal wall to open up the hypopharynx. With impairment of this forward movement of the larynx, the valleculae can readily fill and are not emptied by compression. It may be, therefore, that there is present some neuromuscular inferiority in this group of muscles which will permit easily of its partial or complete dysfunction from nonobvious causes and so give rise to vallecular filling and retention. Diagnosis of the condition can be made only by careful local and general examination, together with thorough roentgenologic investigation. Either examination alone is valueless.

In thirteen of fourteen of the cases of primary vallecular dysphagia, troublesome symptoms disappeared after explanation and reassurance. One patient apparently went from one hospital to another for advice. Frequent gargling with plain water after meals has been recommended, and also repeated drinking of small amounts of water after meals. Removal of the free portion of the epiglottis has been suggested and appears to be a rational mode of treatment if the symptoms are sufficiently severe. In the secondary forms treatment naturally would be directed toward the primary lesion.

FERGUSON, Niagara Falls, N. Y. [ARCH. NEUROL. & PSYCHIAT.]

Miscellaneous

"THE COMMON COLD." DAVID THOMSON and ROBERT THOMSON, Ann. Pickett-Thomson Research Lab. **8**:1 (Dec.) 1932.

This volume, consisting of 738 pages, contains information extracted from more than 2,000 articles, together with a short account of the authors' researches on the subject of the common cold.

Every major item of the subject is covered—the structure and physiology of the nose, the anatomy of the sinuses, the normal bacterial flora, the chemistry of the mouth and saliva, the various claimed causes of colds and the rôles of various micro-organisms. This is a book that is as important to the average practitioner as to the research worker. The effect of chills, bad ventilation, fatigue, diet and clothing are considered in detail and make the volume valuable to every one in the medical field. The complications and sequelae of colds are adequately dealt with, as are the methods of protection, such as vaccines and sprays. There is more information on the common cold packed in this volume than has ever before been collected in one book.

A glance at the publication is sufficient to make one wish to own a copy.

TOOMEY, Cleveland. [AM. J. DIS. CHILD.]

Iodized Oil in Bronchiectasis. Joseph A. Weinberg, Arch. Surg. **27**:545 (Sept.) 1933.

The author makes several deductions as to the fate of iodized oil in bronchography and in the treatment of bronchiectasis. His conclusions are based on a study of two cases following lobectomy and on experiments with iodized oil in animals. Postoperative study of two bronchiectatic human lungs into which injections of iodized oil had been made showed that iodized oil may be retained in the lung for at least eighty-three days following its introduction into the bronchial tract and that most of the cells found in the lung are capable of ingesting droplets of the oil.

Experiments with animals showed that an excessive amount of iodized oil was a factor in the development of pneumonitis. This occurrence and the occurrence of atelectasis in these animals indicate that only small amounts of iodized oil should be used for diagnostic purposes in clinical work.

Kaiser, Rochester, N. Y. [Am. J. Dis. Child.]

Lipiodol in the Diagnosis of Congenital Oesophageal Atresia. F. Fraser Brockington and R. Lightwood, Arch. Dis. Childhood **8**:397 (Dec.) 1933.

Iodized poppy-seed oil 40 per cent was injected into the esophagus in two cases of congenital esophageal atresia. In case 1 roentgenograms taken during life showed a blind end to the esophagus, with a little iodized oil in the stomach and lungs. Postmortem examination revealed complete atresia of the esophagus. There was a communication extending from the trachea to the lower portion of the esophagus.

In case 2 roentgenograms taken ante mortem revealed complete atresia of the esophagus. Postmortem injection of the iodized oil into the trachea and esophagus showed no communication. This condition without communication occurs in only 10 per cent of cases of congenital esophageal atresia. Injection of the iodized oil is a valuable diagnostic aid in esophageal atresia.

Kelly, Milwaukee. [Am. J. Dis. Child.]

On the Nature of High Arched Palate. O'C. O'Sullivan, Brit. M. J. **1**:800 (May 5) 1934.

The author discusses the frequency with which high-arched palate is found in the presence of evidences of insufficiency of vitamin D. He then discusses briefly the mechanical forces which might cause arching of the palate or narrowing of the alveolar arches. He states that in these cases there is little or no encroachment on the antrums and further states that there is no elevation of the palate, the apparent height of the roof being due entirely to abnormal downward elongation of the alveolar process. Development of the roof of the mouth is discussed in order to explain this formation. There are, however, certain types of this condition which are apparently congenital. These, he thinks, are due to persistence of a state of fetal development.

Royster, University, Va. [Am. J. Dis. Child.]

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY, OTOLARYNGOLOGICAL SERVICES

Regular Weekly Meetings, July and August 1934

EDITED BY PHILIP E. MELTZER, M.D.

A CASE OF ACUTE MASTOIDITIS, SEPTICEMIA AND ABSCESS OF THE BRAIN. DR. D. K. JUDD and DR. L. A. SCHALL.

A girl, aged 19, entered the Massachusetts Eye and Ear Infirmary for the first time on March 15, 1934, because of a discharge from the right ear. About four weeks before admission she had a cold and within a few days a discharge from the left ear, which continued for four days and then stopped. Three days before admission she had a discharge from the right ear. This began spontaneously following a chill. For ten days before entering she had a septic temperature and frequent sweating, and one week before coming in, another definite chill. On the day of admission she was operated on. The right jugular vein was ligated, and a simple mastoid operation was done on the right side. At operation a lumbar puncture showed an initial pressure of 180. The Tobey-Ayer test indicated a block on the right. Examination of the spinal fluid gave negative results. The mastoid cells contained purulent secretion. There was not much destruction in the sinus. The sinus itself appeared normal, and free bleeding was obtained from it.

On March 25, the patient was transferred to the Massachusetts General Hospital because of abscesses of the soft tissues which had appeared in the regions of the right elbow and the dorsum of the left foot. The arm was incised and drained, and a hemolytic streptococcus was obtained from the pus. The culture from the right mastoid showed a similar organism.

About one week later the patient became drowsy and confused and showed muscular weakness and loss of sensation of the left hand and arm. A lumbar puncture showed an initial pressure of 400, but the dynamics were normal. The fluid showed 6 cells, all lymphocytes. The patient had between 3 and 4 diopters of papilledema. There was a question of weakness of the left side of the foci. The abdominal reflexes on the left side were gone. There was a positive Oppenheim sign on the left. The Babinski sign was negative. Probable abscess of the right temporal lobe was diagnosed.

The patient was transferred back to the Massachusetts Eye and Ear Infirmary, where a decompression and exploration for abscess were done by Dr. L. A. Schall. The middle and posterior fossae were widely decompressed, and the temporal lobe and cerebellum were explored for an abscess, but none was found. The sinus was reopened, and through this the posterior fossa was explored, but no pus was obtained.

The members of the neurologic service believed that there was an abscess, according to their examinations, and advised transferring the patient back to the Massachusetts General Hospital for exploration higher up. She was transferred back there two days after the exploration here, and Dr. Tracy Putnam, a neurosurgeon, did a decompression of the right parietal region. It was stated in the record that on opening the dura exudate and pus were found on the surface of the brain. I do not think that the exudate amounted to much. I could not see much of anything during the operation, but the brain appeared to be under considerable pressure, and there was thought to be an area of softening in this region. However, exploration with the needle disclosed no abscess and no frank pus. The

dura was tightly closed over the exposed area, and the scalp wound was closed, leaving a decompression wound.

Following this operation the patient's illness took a stormy course, during which marked edema of both eyes developed which spread over the face and neck and down onto the chest. The area over the decompression, in taking up the intracranial pressure, bulged out markedly. On the fifth day following operation the scalp sutures were removed. On the sixth day there was a spontaneous discharge of pus from the area of the decompression.

Since then the patient's temperature has become normal; she looks much better and her mind is clear. Also, she has regained partial muscular control of the left arm. The wound continued to discharge, and on April 10 it was opened somewhat wider and there followed a gush of thick pus. This the neurologists believed to be coming from an abscess of the brain. They did not plan to do anything further at that time. There was still marked swelling over the decompression.

DISCUSSION

DR. L. A. SCHALL: This is the patient whom I saw the last day of the last service. She was transferred from the Massachusetts General Hospital with a question of abscess of the temporosphenoid lobe. I tried to find a stalk connected with the mastoid. I explored the temporosphenoid lobe and the cerebellum and could not find it. The neurologists still insisted that there was an abscess in the parietal lobe. They explored and did not find it. Nature has helped out, and perhaps the patient will get well.

DR. D. K. JUDD (making a further report of the progress on June 20): The large mastoid cavity healed slowly but was nearly closed when the patient was discharged in April. At home her condition remained about the same, with a slight discharge from the decompression wound and slow improvement in the muscular action of the left arm and leg. On the night before readmission here on June 13, however, she had a profuse hemorrhage from the mastoid wound, which was controlled by her local doctor with a pressure bandage. There was a question of whether this bleeding came from the lateral sinus. Following her return to the hospital she had no further bleeding until the night before last, when it was rather brisk, apparently venous, and easily controlled.

A CASE OF CONGENITAL SYPHILIS WITH ACUTE SINUSITIS AND OSTEOMYELITIS OF THE FRONTAL BONE. DR. J. R. FRAZEE, DR. E. H. GILLESPIE and DR. M. H. LURIE.

This is a girl of 13 years with congenital syphilis and left pansinusitis who was admitted to the hospital on April 1, 1934, because of swelling of the eyelids. Thirteen days before admission she caught a cold. There was a profuse watery discharge from her eyes and nose. Five days before admission, when the cold seemed to be improved, the left eyelids became swollen, and following that, the right eyelids. She had no headache, vomiting, dizziness, fever or chills. She appeared comfortable.

Examination showed a typical syphilitic facies with involvement of the base of the nose and a fluctuant area about the root of the nose extending about 1 inch (about 2.54 cm.) upward to the base of the nose and laterally to each eyelid. She did not appear acutely ill. The history obtained was that the mother had had treatment for the past year. Two years ago the patient had involvement of the nose and forehead and a positive Wassermann reaction. She was given arsphenamine, which did not prove very efficacious. A bismuth compound was used, and she seemed to improve considerably with the use of this drug. The last four months she has had treatment only by mouth.

On admission the white blood cell count was 20,000 with 83 per cent polymorphonuclears. The temperature was 101 F., and after that time she had an evening rise of temperature to about 103 F. The dermatologist who was consulted said that her condition was noninfectious, and that she was in good enough state

for operation, if that was desired. The question is: How much of her trouble is due to syphilis and how much to sinusitis?

DISCUSSION

DR. A. S. MACMILLAN: The x-ray photographs show pansinusitis of the left side with involvement of the frontal bone. The osteomyelitis involves the right side of the frontal bone, which is more or less typical of osteomyelitis. The walls of the sinuses are thickened—especially those on the left—the walls of the frontal sinus and the anterior wall over it, all the way up to the skull line. A lateral view shows that the rest of the skull is uninvolved.

DR. H. P. MOSHER: The sinusitis is probably secondary. All the sinuses are involved.

DR. F. E. GARLAND: What would you do in this case?

DR. H. P. MOSHER: Open the subperiosteal abscess and then wait.

DR. C. T. PORTER: The swelling is more fluctuant at the base of the nose. She has a saddleback nose. She must have had a gumma involving the nasal bones and also this area here. I think the sinuses are blocked off now. She has absolutely no drainage. The thing to do is to drain the sinuses, and if any sequestrums form, remove them—just drain through that central area in the bridge of the nose.

DR. F. E. GARLAND: Is there a chance of infection entering the wound?

DR. C. T. PORTER: I shouldn't think so.

DR. E. W. HERMAN: I should be inclined to agree with Dr. Porter on simple drainage. The chance of infection would not be greater than the benefit derived.

DR. L. A. SCHALL: I should put her under antisyphilitic treatment and watch her a few days before operating.

DR. M. H. LURIE: The treatment has been started only within the last forty-eight hours and has not had a chance to show results. She is getting 48 drops of potassium iodide. If the swelling does not increase, I should be inclined to wait.

DR. J. R. FRAZEE (making a further report of the case one week later): Five days ago Dr. Gillespie incised the abscess over the inner canthus of the right eye and extending downward toward the lower limit of the fluctuation about the tip of the nose. Through the wound one could see the extent of the frontal bone and the other side of the nose. The bone appeared bare, rough and irregular, just as in the x-ray photograph. The day after operation the swelling in both eyelids decreased considerably, the edema subsided, and she could open her eyes. On that day the rubber cigaret drain was replaced by three catheters. The treatment with potassium iodide has been continued. She has been comfortable, with no headache. The temperature since the operation five days ago has not reached 100 F.

DR. C. H. ALLMAN: What kind of incision was made?

DR. J. R. FRAZEE: From the inner aspect of the right eyebrow down between the nasal bridge and inner canthus to the bone. Pus was found under pressure, not foul but thick. A culture showed a streptococcus and the influenza bacillus.

DR. M. H. LURIE: There is no reason why she should not have an uneventful convalescence without further intervention.

A CASE OF ABSCESS OF THE BRAIN. DR. L. A. SCHALL.

Many of you remember my report last year on a 14 year old boy with a history of spontaneous rupture of the drum three weeks prior to examination, a discharge from the left ear for two weeks and a dry ear for one week, with increasing headache. He was admitted on April 28, 1933, when he had a stiff neck, a high temperature, a Babinski sign—all the signs of meningitis. An x-ray photograph showed destruction of the left mastoid. The treatment was a simple mastoidectomy.

The mastoid was found broken down and filled with pus. The dura over the antrum, mastoid and squama was exposed. The dura appeared to be normal; it was incised and a drain inserted between the dura and the brain. Lumbar puncture showed a spinal fluid pressure of 450; the fluid was turbid, with 7,500 cells. Pneumococcus type I was found on smears and on lumbar puncture. The treatment consisted of intraspinal administration of antipneumococcic serum type I, reinforced by human complement as well as by the serum given intravenously. This boy got well.

He remained well until 1:30 a. m. Sunday, April 8, 1934, three days ago. He complained of earache. He was given acetylsalicylic acid. In the morning he had no pain. He ate breakfast, and in the middle of this meal he showed sudden aphasia. He could not name the things he desired. This lasted from five to ten minutes; then he had a convulsion and became unconscious. He was brought in here unconscious Monday morning.

The neurologic findings showed weakness of the right side of the face and somewhat diminished reflexes on the right side. The eyegrounds were normal. The temperature was 100 F. The probable diagnosis from the history was abscess of the left temporosphenoid lobe.

An exploration was done. The old mastoid incision was reopened. The dura over the former decompression was very tense. The mastoid cavity was filled with what looked like herniated brain tissue, black, necrotic. I could feel no dura over the mastoid portion. The dura over the squama was intact, but the dura over the mastoid seemed to be absent. In fact, I could fit my finger up into this necrotic area of the brain as if going up through the mastoid. The area was sterilized with diathermy; then with the Cushing needle I explored the temporosphenoid lobe without finding an abscess. I cleaned out the necrotic brain tissue which protruded into the old mastoid cavity and inserted a soft rubber drain.

The next morning the patient was rational and continued to be rational. He had no aphasia, no headache and no other symptoms save a slight nausea, which may be significant of something else. I have not done a lumbar puncture, because he has no meningeal signs.

DISCUSSION

DR. M. H. LURIE: From the evidence the condition seems to be a cortical abscess rather than an abscess of the temporosphenoid lobe.

DR. H. P. MOSHER: How many cases of this character have been seen here?

DR. M. H. LURIE: It is the first one which I have seen.

DR. H. P. MOSHER: I have never seen a case like this. The only thing I can think of is that if Dr. Schall had known or felt that this was all there was to it, he would not have put his needle up farther. That may cause him trouble, but I think he will be forgiven for that.

DR. D. C. SMYTH: I was going to ask Dr. Schall where he did needle.

DR. L. A. SCHALL: I needled first up through the black necrotic area and then I needled up above.

DR. D. C. SMYTH: What was the history in the interval? It seems to me that he had a localized meningitis which formed a localized subdural abscess.

DR. L. A. SCHALL: He has had absolutely no symptoms. He has gained in weight. He has been getting on in school. He has had no headache.

DR. D. C. SMYTH: It is an unusual case. Did he have a dry middle ear all along?

DR. L. A. SCHALL: Yes, sir. The examination of the drum showed just a little congestion around the periphery.

DR. G. L. TOBEY: Did you take a culture of this mass?

DR. L. A. SCHALL: There was no pus to take it from. I do not think he is out of danger yet.

NOTE.—On November 20, the patient was reported on as being perfecly well.

A Case of Foreign Body in a Bronchus. Dr. J. R. Frazee and Dr. C. T. Porter.

A boy was admitted four days ago because of a foreign body in the chest. About six days ago, while coming home from school, he was playing with a metal eraser cap of a pencil. He was whistling on it and got it down his throat. He was taken to an outside hospital, where x-ray photographs showed the foreign body to be at the level of the sternum and at the level of the sixth or seventh rib posteriorly. He was given cathartics. An x-ray photograph indicated that the foreign body did not move. An otolaryngologist was told about the patient, and he sent the boy here.

Another x-ray photograph showed the foreign body about as described previously. Dr. Porter operated on the boy and removed the foreign body. Since the operation the patient has done well. He has had practically no reaction, but has a little dulness and a few wheezes on that side of the chest. An x-ray photograph taken this morning shows little congestion.

DISCUSSION

Dr. C. T. Porter: This foreign body pointed down, according to the x-ray photograph. It had been in the boy since Thursday. I saw him Sunday. It had gone down as far as it could go in the left main bronchus. The edges were completely obscured by swollen edematous bronchial mucous membrane. Finally I got the edges partly freed and got the staple spreader in there. I could not get hold of the edges with the grasping forceps. I tried twice to free it by grasping and then I simply put this forceps down in there and spread it and after three tries the cap became loose and I got it up. It was 9 mm. in width.

Dr. H. P. Mosher: That was prettily done. Dr. Porter asked for a pair of forceps of the spreading type with teeth on the side so that with a foreign body of this nature we should have a pair of forceps which would grasp it. We had a pair of forceps made along the lines suggested by Dr. Porter. I think we have had three patients of this type admitted in a moderately short time. What bothers me is why, with an x-ray photograph like this and a foreign body away over to the left, the patient should have been given mineral oil.

A Case of Lupus of the Nose. Dr. J. R. Frazee and Dr. M. G. Evans.

This woman, 24 years old, was admitted to the Massachusetts Eye and Ear Infirmary two years ago following pansinusitis, and at that time it was noticed that there was a small papillomatous mass on the right side of the septum and the anterior tip of the left inferior turbinate. A specimen was taken at the time and showed typical lupus vulgaris. Since that time she has had no treatment except for eczema. The patient was readmitted for study. This slide is from the specimen taken two years ago. It shows definite tubercles with central necrosis and a peripheral granulomatous reaction with giant cells.

There is nothing suggestive of acid-fast bacteria in the history except abscesses in the right side of the neck which were opened when the patient was 12, and she was given radiation treatment. At present, the only complaint is of nasal obstruction from the mass, which has continued to enlarge and now obstructs both anterior nares. Only since last October has it spread to the outer skin over the tip of the nose. An x-ray photograph of the sinuses is normal. The textbooks state that at times there is involvement of the sinuses, but in this patient they are clear. Dermatologists were consulted. They are much interested in this type of lupus, having treated many patients by diet alone. The diet is salt-free, the salt being replaced by a substitute which contains no sodium chloride.

Dr. M. G. Evans: I saw this patient two years ago, and at that time she had acute pansinusitis of the left side. With all the pus in the left side of the nose, I noticed a small area of moth-eaten appearance on the anterior part of the septum. One week later, there was a similar area on the anterior tip of the turbinate. I sent her here and a biopsy was done, which showed lupus. I warned

her against a possible spread of the infection, but apparently she did not heed the warning, and I did not see her again until last October. None of the skin of the nose was involved last October. Apparently the disorder started to spread from then on, and for the past six months she has been treated for eczema of the nose by a local doctor.

A Case of Chronic Suppurative Otitis Media and Abscess of the Left Temporal Lobe. Dr. D. K. Judd and Dr. L. A. Schall.

A man, 38 years of age, entered this infirmary on April 13, 1934, complaining that he had had convulsions on the two preceding nights. He stated that at the age of 10 years he had had a discharge from both ears which lasted several months. From that time until the present he had had no trouble except a questionable slight discharge from the ears off and on.

Three weeks prior to admission he began having a discharge from the left ear and one week later from the right ear. There had been no head cold or other illness at the time the discharge began. His wife stated that for several months she had noticed a slight change in his personality, but had not thought much about it. She could describe it only as a tendency toward listlessness and inattention. He had no headaches, no dizziness and no vomiting.

Two nights prior to admission he had had a generalized convulsion—not starting on one side or the other, so far as his wife could remember. After the convulsion he remained unconscious for about three hours. He was brought to the emergency ward of the Massachusetts General Hospital, where a general medical and neurologic examination was made but nothing found. Lumbar puncture showed an initial pressure of 120; the fluid showed 740 cells, mostly polymorphonuclears. Because of the discharge from the ears, he was transferred to the infirmary.

Examination showed a large marginal posterior perforation in each ear, with moderate purulent discharge, but no other abnormality. X-ray photographs showed both mastoids sclerosed, with no enlargement of the antrums. A tentative diagnosis of a localized area of meningitis was made, but since there was no aphasia, difference in the two ears or localizing signs, it was decided to await developments. Another lumbar puncture showed an initial pressure of 150 and 240 cells, 47 per cent of which were polymorphonuclears. The fields and fundi and the neurologic findings were normal.

Three days after admission aphasia was noted, and Dr. Solomon of the neurologic service was asked to see the patient. He found definite aphasia and made a diagnosis of abscess of the left temporal lobe. A few hours later a radical mastoid operation on the left and a decompression of the middle fossa were done by Dr. Schall. The mastoid was extremely sclerosed. The antrum was not enlarged, and there was no cholesteatoma. The tegmen was intact and hard, but was removed over the middle ear, antrum and mastoid. The squama was removed upward for about half an inch (about 1.27 cm.). The dura appeared to be under slightly increased pressure. There was a suspicious area over the antrum. The dura was sterilized with diathermy and a Cushing needle introduced into the temporal lobe. At a depth of about 1 cm., foul brownish pus was found. With the diathermy knife a circular area of the cortex was removed down to the abscess cavity, which had a definite capsule. A Mosher basket drain was inserted. After the operation the patient seemed quite well. He had no complaints. The pulse rate was about 60 and he still had aphasia, but aside from that the examination showed no irregularities. There continued to be a moderate discharge through the basket drain. The culture yielded a staphylococcus.

On the morning of the ninth postoperative day, however, the patient had another convulsion, which was generalized, ushered in by a cry and accompanied by cyanosis and loss of sphincter control. There were no localizing signs. Following this he was stuporous for about thirty minutes. After that the aphasia was found to be practically complete, especially the motor aphasia. At first there was muscular weakness of the right side, but this disappeared within two hours.

That afternoon he was operated on again. The basket drain was removed, and by digital examination the cavity of the abscess was found to extend backward about 4 cm. from the drain; i. e., the drain had been placed close to the anterior limits of the abscess.

The operative field was resterilized and a decompression made through the posterior parietal bone. The cavity of the abscess was located with the Cushing needle, opened with diathermy and a Mosher basket inserted. Another basket was then reinserted through the mastoid wound.

After that operation, for one or two days the aphasia continued to be almost complete, and he was unable to say anything coherently. On the third day he showed definite improvement, although the pulse rate was between 50 and 60. The aphasia gradually cleared until on May 2, eight days after operation, he could tell the names of most simple objects. There was still a brownish discharge from both drains. An attempt was made to inject iodized poppy-seed oil 40 per cent for roentgen studies of the size of the cavity.

DISCUSSION

Dr. A. S. MacMillan: On the x-ray plate the patient does not show much. It might be that the cavities are collapsed. If the iodized oil had gone into any sort of cavity it would have remained there.

Dr. H. P. Cahill: It is one of the most satisfactory cases ever presented here. I feel that Dr. MacMillan has obliterated that cavity. There would otherwise be some little place where that oil would remain. The operator should be congratulated for what he has accomplished and what he dared to do.

Dr. H. P. Mosher: I have never seen a case in which two basket drains were used. The second point is in connection with the second operation: The neurologists said to go forward rather than backward. Dr. Schall put his finger into the first cavity and felt the abscess going backward instead of forward. To that extent it is hard even for a neurologist to be sure about such conditions.

Dr. L. A. Schall: I feel that the cavity is collapsed. We put the iodized oil in with a medicine dropper.

Dr. D. K. Judd (making a further report on the case, May 16, two weeks later): After the decompression and insertion of the Mosher basket drain into the posterior part of the cavity, the patient improved, and there was a discharge from both drains until about two weeks ago, when that from the drain through the mastoid cavity stopped. After injection of iodized oil, x-ray photographs showed that the cavity was probably almost collapsed; the lower drain was then removed, but the wound was kept open. The patient has been up and walking around, with no complaints, but still had a discharge through the posterior drain until a few days ago. The pulse rate is practically normal. The aphasia is present but not so marked; i. e., he is able to name most ordinary objects, although it sometimes takes him several minutes to think of their names. It is planned to remove the remaining Mosher drain this afternoon.

Note.—The patient was finally discharged to the outpatient department.

A Case of a Retro-Esophageal Mass. Dr. J. R. Frazee and Dr. M. H. Lurie.

This man, aged 66, was admitted to the hospital about three and one-half weeks ago with a story of sore throat, difficulty and some pain in swallowing for three months, hoarseness for six weeks and increase in the difficulty of swallowing for about a month. He had redness and swelling of the right arytenoid and right aryepiglottic fold, overhanging the true cord. The left cord moved freely. A 3 mm. ulceration, whitish, was seen on the posterior lateral aspect of the right side of the hard palate. A lateral x-ray plate of the neck showed a large mass (smooth) behind the upper end of the esophagus. Barium sulphate passed freely over this mass and was deviated anteriorly to the left.

With the patient under local anesthesia, Dr. Evans did a tracheotomy and then with the Jackson laryngoscope looked down from above and saw behind the right arytenoid what he thought was a small outcropping. A 12 inch (30.48 cm.) Mosher esophagoscope was introduced, and the mass was seen to extend down behind the cricoid. With biting forceps two specimens were taken from the retro-arytenoid space. A biopsy showed questionable hemangioma.

About two weeks ago Dr. Lurie passed the esophagoscope under ether, which showed this rounded mass in the right posterior esophageal wall at the level of the cricoid. It looked so much like an abscess that he incised it and had the sensation of thrusting into a cavity. Neither pus nor free bleeding was obtained. Specimens were taken which showed no evidence of malignancy.

Six days ago Dr. Lurie made a 5 inch (12.7 cm.) incision along the anterior border of the right sternomastoid muscle extending up to the level of the hyoid. The sternomastoid was retracted laterally and blunt dissection carried through the deep cervical fascia. The anterior belly of the digastric muscle was cut. The carotid sheath and contents were retracted laterally. The pharyngeal constrictors were retracted after blunt dissection. The large right upper pole of the thyroid gland was pulled forward, and the dissecting finger came on a rather firm irregular mass which could not be dissected out without breaking into a cavity. No further attempt to remove this was made. Specimens were taken. A cigaret drain was placed in the cavity.

Four days after operation, the wound was all healed except a small portion at the lower end from which there was some drainage. The patient had little reaction from the operation. I forgot to mention a stomach tube which was inserted by direct view at the time of the last operation.

DISCUSSION

DR. M. H. LURIE: There were two drains, one running in through the region of the esophagus and the other underneath the subcutaneous tissue, but the latter came out in forty-eight hours.

DR. J. R. FRAZEE: The last biopsy showed chronic inflammatory with some perivascular inflammation, but no evidence of malignancy.

The Wassermann and Hinton tests were negative.

A NASAL FRACTURE THE RESULT OF DIVING. DR. BURTON E. LOVESEY AND DR. V. H. KAZANJIAN.

A young man, about ten days before admission to the hospital, was standing on a springboard facing the water; he jumped, making a reverse circle, and struck his nose on the springboard. He was taken to a hospital in Lawrence. He was not knocked unconscious. At that hospital he was treated conservatively and then sent here for a plastic operation. Examination showed the nose much flattened and the septum crushed, in mushroom fashion, downward onto the floor. The patient was taken to the operating room, and Dr. Kazanjian used a pair of forceps with long blades, inserting them into the nose, and with a slow steady upward pull was able to bring the flattened nose and septum into a good position. Two finger cots were then placed in each nostril. A splint of dental composition and metal was then molded to the nose with the outside part attached to a frame. One part of the splint was embedded in dental compound, placed in the nose and wire inserted. Then another piece of dental compound was placed on the forehead and a groove director bent down in that fashion. Elastics were applied to keep the nose suspended. This was kept on apparently for from four to five days.

DISCUSSION

DR. V. H. KAZANJIAN: It is not necessary to keep the splint on more than from four to five days. The septum was really crushed and flattened, and we were unable to see inside the nose for ten days. I was not sure whether or not we could raise it with counterpressure and hold it in position.

TWO CASES OF MULTIPLE FRACTURE OF THE NASAL AND FRONTAL BONES. DR. BURTON E. LOVESEY and DR. V. H. KAZANJIAN.

DR. B. E. LOVESEY: A man, 22 years of age, on June 10, 1934, was in a fistic encounter and was severely beaten about the face. Examination in the emergency ward showed moderate epistaxis with expectoration of blood, which probably came from the nose. He had headache and some vertigo, and vomited blood several times. There was nothing noteworthy in the past history.

On June 12, he still had frontal headache.

On June 15, he was transferred to the plastic service, and the next day he was operated on. Dr. Kazanjian made an incision supra-orbitally ¾ inch (1.91 cm.) from the eyebrow. With an elevator the fracture line through the frontal bone was found, and by gradual pressure the nasal bone, frontal bone and left supra-orbital margin were elevated. The nasal bones were then reduced and shaped into position. A drill hole was made in the frontal portion of the elevated bone, and a brass wire passed through this and through the fracture line and brought outside of the skin and twisted together.

Four or five days later, a dental composition and wire splint were applied. When the frame was in place, an elastic band was attached to it and the brass wire. The patient was quite comfortable and was allowed to go home. He returns today for inspection.

DR. V. H. KAZANJIAN: The second case was a little more extensive. A patient at the Baker Memorial Hospital had a bad fracture of the nasal bone. There was a considerable amount of comminution. An incision was made below the eyebrow, and the frontal sinus was exposed. We found small fragments of bone scattered in the sinus, and they were removed. Then there were two more small pieces of bone, very loose, pressed against the inner wall, and the same method was used; that is, a little hole was drilled through the bone, a wire passed through it, the wire coming through the skin and forming a loop, and then we used the suspension method. It is easy to remove the wire by cutting one end. It leaves no bad effect. One must do the same thing in correcting fractures of the malar bone. One has no control in any other way.

A CASE OF LARYNGOFISSURE. DR. P. E. IRELAND and DR. PHILIP MYSEL.

A man 48 years old had been hoarse for one a half years. He had no other symptoms, no pain and no difficulty in breathing. He said that four months prior to examination a biopsy was made in a Connecticut hospital and laryngectomy advised. An x-ray plate of the chest and a Wassermann test were negative. On examination on July 2, 1934, there was a nodular mass on the anterior third of the right cord. The arytenoid was not fixed on that side. The mass extended to the anterior commissure. Nothing could be seen on examination of the other cord. A biopsy was reported as showing active epithelial hyperplasia, probably malignant. There was not sufficient tissue to show infiltration, and a positive diagnosis was not made. The patient was let go, and he came back ten days later. Examination then showed the same condition.

Dr. Schall requested a second specimen for biopsy, and this time it was reported as "rapidly growing atypical papilloma. No definite evidence of a malignant growth can be made out."

On July 21, Dr. Schall advised splitting the larynx and removal of the tumor. Dr. Mallory thought that the condition might be malignant but was not sure; he felt at that time that a laryngofissure should be made anyway. So, under local anesthesia, Dr. Mysel split the larynx, and it was found that the region involved was mostly confined to the anterior third of the right cord, but that there was a suspicious area on the anterior tip of the left cord. All the tissue was dissected free from the cartilage on the right. It dissected readily. An incision was made above the ventricular band back toward the arytenoid and down to the arytenoid on that side, and the whole cord and ventricular band with about 0.5 cm. of subglottic tissue were removed from the anterior commissure back to the

arytenoid, including the vocal process. This was done with the diathermy snare. At the same time the anterior third on the left side was removed. The remainder of the tissues in the arytenoid region was left in place. The wound was sewed up tightly. The feeding tube was not used. The patient was a little uncomfortable that afternoon but is comfortable now. The temperature was 102 F. that night but is now normal.

DISCUSSION

DR. P. MYSEL: This patient had a growth on the right side extending from the commissure to the vocal process and also subglottically. On the left side the growth extended from the midportion of the cord to the commissure. On the right side I did a complete resection, taking the ventricular band, the cord and the subglottic area, including a part of the vocal process. On the left I did the same but left the posterior half of the cord.

DR. D. C. SMYTH: Did you sew the wound up?

DR. P. MYSEL: Yes, but the patient had a tracheotomy tube. Although the wound was perfectly dry following operation, it began to leak from above in the afternoon.

DR. P. E. MELTZER: What is going to be the outcome now that both cords are denuded?

DR. P. MYSEL: Granulations will fill up the area that has been removed, and eventually new epithelium will cover the denuded areas.

DR. P. E. MELTZER: I thought when the cords were denuded on both sides, granulations would spring up and the result would be stenosis.

DR. P. MYSEL: No; that is, not so as far as stenosis is concerned. Sir St. Clair Thomson has reported 3 cases in which both sides were denuded without stenosis to cause interference in airway.

DR. D. C. SMYTH: In reply to Dr. Meltzer, I should like to say that the arytenoids are moving; the thyroid cartilages are present. Granulations will form and disappear. Looking at this patient a year from now you will hardly know that you removed the cords, as new cords develop from the scar tissue.

Book Reviews

Annals of Roentgenology. Volume XV. Nasal Accessory Sinuses Roentgenologically Considered. By Frederick M. Law, M.D. Edited by James T. Case, M.D. Price, $10. Pp. 215, with 197 roentgenologic studies, 28 clinical illustrations and 3 key plates. New York: Paul B. Hoeber, Inc., 1933.

This volume includes a chapter on the anatomy of the nasal accessory sinuses, roentgenologically considered. The variations in the structure of the paranasal sinuses are so conspicuous that any one who undertakes to evaluate the clinical or roentgenologic findings must have a proper appreciation of the structural variations. The chapter on anatomy is followed by one on general technic which, on the whole, is more difficult in the study of roentgenologic findings in the sinuses than in the mastoid. Many puzzling questions of interpretation are explained in the light of the author's wide experience.

In discussing technic the author defines and illustrates angles for the optimum approach of the various accessory sinuses using but little special apparatus and argues against the use of grid diaphragms. The text includes more than the usual emphasis on diagnostic points attainable in the ethmoid region from stereoscopic lateral views.

The volume is well illustrated, containing, in all, 225 figures, mostly reproductions of roentgenograms. The book approaches perfection from the standpoint of good printing and book making.

A review of volume I of this series, on the mastoids, by the same author, was published in the ARCHIVES (**10**:711 [Dec.] 1929).

Sculpture in the Living: Rebuilding the Face and Form by Plastic Surgery. By Jacques W. Maliniak, M.D. With a foreword by Wendell C. Phillips, M.D. Price, $3. Pp. 203. New York: The Lancet Press, 1934.

This is not a scientific contribution to plastic surgery. It is a chatty discussion of plastic surgery of the head and face. It is written in a light vein and is intended more for the edification of the lay public than for the guidance of surgeons interested in correcting facial deformities. Scattered through the volume are many suggestions which should be appreciated by the lay public, especially those regarding the pitfalls laid for persons who are inclined to seek aid from quack "beauty doctors" for real or imaginary difficulties. The physician engaged in general practice will find in it the answer to many questions regarding facial deformities which may be put to him by patients.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143, Groningen, The Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John J. Shea, 1918 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch; 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cincinnati. Time: Sept. 14-20, 1935.

American Bronchoscopic Society

President: Dr. Henryq Hall Forbes, 40 E. 61st St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Toronto, Canada. Time: June 1, 1935.

American Laryngological Association

President: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.
Place: Toronto, Canada. Time: May 29-31, 1935.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Perry G. Goldsmith, 170 St. George St., Toronto, Canada.
Secretary: Dr. R. L. Loughran, Sharon, Conn.
Place: Toronto, Canada. Time: June 3-5, 1935.

SECTIONS:

Eastern.—Chairman: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Southern.—Chairman: Dr. Beverly R. Kennon, Medical Arts Bldg., Norfolk, Va.
Middle.—Chairman: Dr. Daniel W. Layman, Medical Arts Bldg., Indianapolis.
Mid-Western.—Chairman: Dr. Edward H. Cary, Medical Arts Bldg., Dallas, Texas.
Western.—Chairman: Dr. Frank A. Burton, Bank of America Bldg., San Diego, Calif.

American Otological Society

President: Dr. S. J. Crowe, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Toronto, Canada. Time: May 27-29, 1935.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.